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Effects of Pressure Control Versus Volume Control Assisted Ventilation on Patient Work of Breathing

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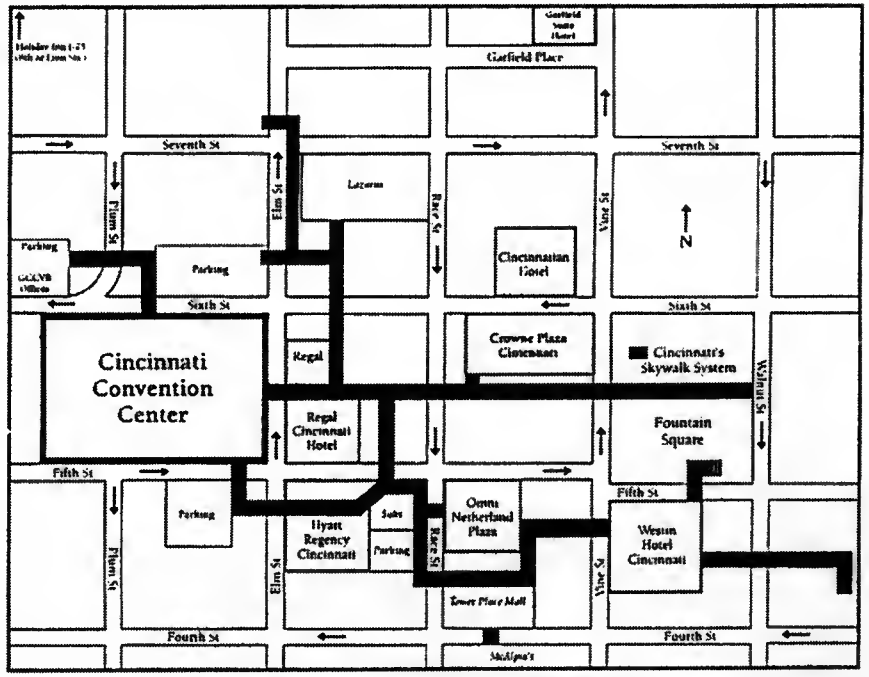

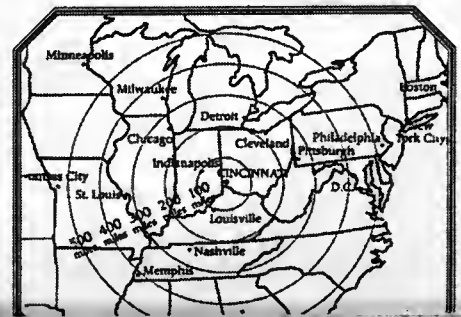

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RESPIRATORY CARE

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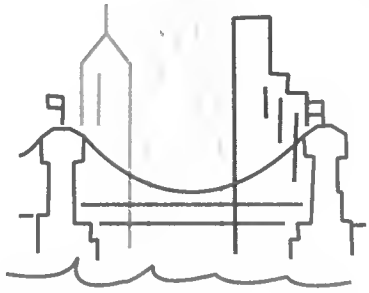

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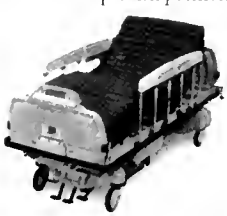

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Idiopathic Pulmonary Fibrosis: Diagnosis and Treatment: International Consensus Statement—American Thoracic Society (ATS) and the European Respiratory Society (ERS). Am J Respir Crit Care Med 2000;161:646–664.

Do Long-Acting Beta2-Adrenergic Agonists Deserve a Different Place in Guidelines for the Treatment of Asthma and COPD?—van Schayck CP. Eur Respir J 2000;15(4):631–634.

"The Skeletal Muscle in Chronic Respiratory Diseases." Summary of the ERS Research Seminar in Rome, Italy, February 11–12 1999—Palange P, Wagner PD. Eur Respir J 2000; 15:807–815.

Relation of FEV_1 to Clinical Outcomes during Exacerbations of Chronic Obstructive Pulmonary Disease —Department of Veterans Affairs Cooperative Study Group. Niewoehner DE, Collins D, Erbland ML. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1201-1205.

FEV_1 is an objective measure of airflow obstruction used in clinical practice and in therapeutic trials. The precise relationship of FEV_1 to clinical outcomes is generally uncertain. As part of a randomized trial to assess systemic corticosteroid efficacy, we obtained serial FEV_1 measurements in patients hospitalized for exacerbations of chronic obstructive pulmonary disease (COPD). Over the first 14 Study Days at least one FEV_1 value was obtained in 261 subjects. Sixty-four of these subjects experienced treatment failure, defined as death, intubation, readmission for COPD, or intensification of drug therapy, by Study Day 30. After adjustment, both FEV_1 at entry into the study (odds ratio [OR] for a 100-mL increase, 0.87; 95% confidence interval [CI], 0.79 to 0.96) and change in FEV_1 over the first two Study Days (OR for a 100 mL increase, 0.80; 95% CI, 0.69 to 0.92) predicted treatment failure. We identified no baseline characteristic that was significantly related to FEV_1 at entry into the study. Assignment to the systemic corticosteroid treatment arm was associated with a significantly larger FEV_1 at Study Day two ($p = 0.01$). We conclude that FEV_1 measurements at admission and over the first several days of hospitalization are highly predictive of clinical outcomes during exacerbations of COPD.

Physiologic Determinants of Ventilator Dependence in Long-Term Mechanically Ventilated Patients—Purro A, Appendini L, De Gaetano A, Gudjonsdottir M, Donner CF, Rossi A. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1115-1123.

To investigate the pathophysiologic mechanisms of ventilator dependence, we took physiologic measurements in 28 patients with COPD and 11 postcardiac surgery (PCS) patients receiving long-term mechanical ventilation during a spontaneous breathing trial, and in 20 stable, spontaneously breathing patients matched for age and disease. After 40 ± 14 min of spontaneous breathing, 20 of 28 patients with COPD and all 11 PCS patients were judged ventilator-dependent (VD). We found that in the 31 VD patients tidal volume was low (V_T: 0.36 ± 0.12 and 0.31 ± 0.08 L for COPD and PCS, respectively), neuromuscular drive was high ($P_{0.1}$: 5.6 ± 1. 6 and 3.9 ± 1.9 cm H_2O), inspiratory muscle strength was reduced (Pdi_{max}: 42 ± 12 and 28 ± 15 cm H_2O), and lung mechanics were abnormal, particularly PEEPi (5.9 ± 3.0 cm H_2O) and lung resistance (22.2 ± 9.2 cm H_2O/L/s) in COPD. The load/capacity balance was altered (Pdi/Pdi_{max} and $Ppl/Ppl_{max} > 0.4$) and the effective inspiratory impedance was high ($P_{0.1}/V_T/TI \geq 10$ cm H_2O/L/s). Failure to wean

occurred in patients with $f/V_T > 105$ breaths/min/L and 56% of patients with COPD with $f/V_T < 80$ breaths/min/L. Those who failed despite a low f/V_T (< 80 breaths/min/L) either showed ineffective inspiratory efforts, which artificially lowered f/V_T (n = 8), or did not increase breathing frequency (n = 5), but $P_{0.1}$ and $P_{0.1}/V_T/TI$ were as high as in other VD patients. In the 31 VD patients, P_{aCO_2} increased during the weaning trial (+12.3 ± 8.0 mm Hg). We conclude that in the presence of a high drive to breathe, the imbalance between increased work load and reduced inspiratory muscle strength causes respiratory distress and CO_2 retention. Noninvasive measurements (breathing pattern, $P_{0.1}$, $P_{0.1}/V_T/TI$) may give better insight into weaning failure useful in clinical decision-making, particularly in patients with COPD not showing rapid shallow breathing (56% in this study).

Respiratory Function in Survivors of the United Kingdom Extracorporeal Membrane Oxygenation Trial—Beardsmore C, Dundas I, Poole K, Enock K, Stocks J. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1129-1135

Extracorporeal membrane oxygenation (ECMO) improves survival in mature neonates with reversible lung disease. However, ECMO could result in survival of infants with severe respiratory dysfunction who would otherwise have died. Alternatively, infants receiving ECMO might be spared prolonged ventilation and consequent barotrauma, resulting in improved respiratory function. Our aim was to compare respiratory function at 1 yr of age in infants assigned to receive either ECMO or conventional management (CM). Seventy-eight surviving infants of the United Kingdom (UK) ECMO trial (51 in the ECMO group) were studied at 1 yr of age. Questionnaires provided details of respiratory symptoms, and laboratory measurements of respiratory function were made for respiratory rate, tidal volume, lung volume, airway conductance, specific airway conductance, and maximal expiratory flow at FRC ($\dot{V}_{max}$ (FRC)). Data were exchanged on floppy disk for cross-analysis and to ensure that investigators were blinded to the status of the infants. There was a wide spectrum of respiratory function, from normal to markedly abnormal. There were few differences between the groups, but in the CM group lung volume was increased (95% confidence intervals [CIs] of the difference in ECMO versus CM subjects: -67; -4 mL), and inspiratory specific conductance was lower (95% CI: 0.03; 0.98 $s^{-1} \cdot kPa^{-1}$). There was a trend toward a lower $\dot{V}_{max}$(FRC) (95% CI: -2; 67 mL/s^{-1} in the CM group. In addition to providing a survival advantage, ECMO did not worsen lung function in infants assigned to receive it. Indeed, their lung function appeared slightly better than that of infants treated conventionally.

The Treatment of Acidosis in Acute Lung Injury with Tris-Hydroxymethyl Aminomethane (THAM)—Kallet RH, Jasmer RM, Luce JM, Lin LH, Marks JD. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1149-1153.

Mechanical hyperventilation of acidemic patients with acute lung injury (ALI) requires the use of high volumes and pressures that may worsen lung injury. However, permissive hypercapnia in the presence of shock, metabolic acidosis, and multi-organ system dysfunction may compromise normal cellular function. Tris-hydroxymethyl aminomethane (THAM) may be an effective method to control acidosis in this circumstance. Protonated THAM is excreted by the kidneys, so that carbon dioxide production is not raised. In an uncontrolled study, we administered THAM to 10 patients with acidosis (mean pH = 7.14) and ALI (mean lung injury score = 3.28) in whom adequate control of arterial pH could not be maintained during either eucapnic ventilation or permissive hypercapnia ventilation. THAM was given at a mean dose of 0.55 mmol/kg/h. Administration of THAM was associated with significant improvements in arterial pH and base deficit, and a decrease in arterial carbon dioxide tension that could not be fully accounted for by ventilation.

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Although further studies are needed to confirm these observations, THAM appears to be an effective alternative to sodium bicarbonate for treating acidosis during ALI.

Clinical Evaluation of a Computer-Controlled Pressure Support Mode—Dojat M, Harf A, Touchard D, Lemaire F, Brochard L. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1161-1166.

We have designed a computerized system providing closed-loop control of the level of pressure support ventilation (PSV). The system sets itself at the lowest level of PSV that maintains respiratory rate (RR), tidal volume (V_T), and end-tidal CO_2 pressure (P_{ETCO_2}) within predetermined ranges defining acceptable ventilation (i.e., $12 < RR < 28$ cycles/min, $V_T > 300$ mL [> 250 if weight < 55 kg], and $P_{ETCO_2} < 55$ mm Hg [< 65 mm Hg if chronic CO_2 retention]). Ten patients received computer-controlled (automatic) PSV and physician-controlled (standard) PSV, in random order, during 24 h for each mode. An estimation of occlusion pressure ($P_{0.1}$) was recorded continuously. The average time spent with acceptable ventilation as previously defined was 66 ± 24% of the total ventilation time with standard PSV versus 93 ± 8% with automatic PSV ($p < 0.05$), whereas the level of PSV was similar during the two periods (17 ± 4 cm H_2O versus 19 ± 6 cm H_2O). The time spent with an estimated $P_{0.1}$ above 4 cm H_2O was 34 ± 35% of the standard PSV time versus only 11 ± 17% of the automatic PSV time ($p < 0.01$). Automatic PSV increased the time spent within desired ventilation parameter ranges and apparently reduced periods of excessive workload.

Noninvasive Ventilation with Helium-Oxygen in Acute Exacerbations of Chronic Obstructive Pulmonary Disease—Jaber S, Fodil R, Carlucci A, Boussarsar M, Pigeot J, Lemaire F, et al. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1191-1200.

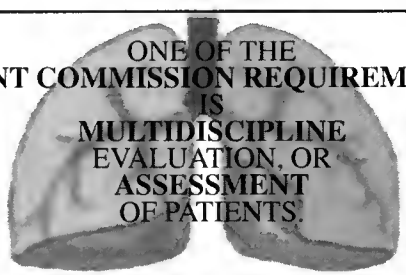
The use of helium-oxygen (HeO_2) was tested in combination with noninvasive ventilation (NIV) in 10 patients with acute exacerbation of chronic obstructive pulmonary disease (COPD). Effort to breathe as assessed by the respiratory muscle pressure-time index (PTI), work of breathing (WOB), and gas exchange were the main endpoints. Results of NIV-HeO_2 were compared with those obtained with standard NIV ($AirO_2$), at two levels of pressure-support ventilation (PSV), 9 ± 2 cm H_2O and 18 ± 3 cm H_2O. Significant reductions in PTI were observed between HeO_2 and $AirO_2$ at both the low PSV level (n = 9; 160 ± 58 versus 198 ± 78 cm H_2O/s/ min; $p < 0.05$) and the high PSV level (n = 10; 100 ± 45 versus 150 ± 82 cm H_2O/s/min; $p < 0.01$). WOB also differed significantly between HeO_2 and $AirO_2$ (7.8 ± 4.1 versus 10.9 ± 6.1 J/min at the low PSV level, $p < 0.05$; and 5.7 ± 3.3 versus 9.2 ± 5. J/min, $p < 0.01$ at the high PSV level). HeO_2 reduced P_{aCO_2} at both the low PSV level (61 ± 13 versus 64 ± 15 mm Hg; $p < 0.05$) and the high PSV level (56 ± 13 versus 58 ± 14 mm Hg; $p < 0.05$), without significantly changing breathing pattern or oxygenation. We conclude that use of HeO_2 during NIV markedly enhances the ability of NIV to reduce patient effort and to improve gas exchange.

Quality Control of Spirometry in the Elderly. The SA.R.A. Study. Salute Respiration Nell'anziano = Respiratory Health in the Elderly—Bellia V, Pistelli R, Catalano F, Antonelli-Incalzi R, Grassi V, Melillo G, et al. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1094-1100.

We evaluated the outcome of the spirometry quality control program of the SA.R.A. multicenter project, the aim of which is the multidimensional assessment of asthma and COPD in the elderly (≥ 65 yr). The factors determining this quality were also evaluated. The program was based on standardized procedures (ATS recommendations), performed by specifically trained and certified personnel; a fully-computerized spirometer with customized software was used for spirometry. A reference center made monthly controls. Overall, 638 cases and 984 controls were examined. Spirometric measurements were obtained in 607 cases and 912 controls; 508 and 747 tests with at least three acceptable curves were obtained in cases and in controls, respectively (NS). The percentage of reproducible tests ranged between 95.8% for FEV_1 in controls and 87.6% for FVC in cases. The average reproducibility for FEV_1 was 61.6 mL in cases and 58.3 mL in controls (NS). Cognitive impairment, shorter 6-min walk distance, and lower educational level were found to be independent risk factors for a poorer acceptability rate (logistic regression analysis). Male sex and age were risk factors for a poorer reproducibility of FEV_1. Reproducibility tended to improve with time ($p < 0.001$). Although spirometry becomes increasingly difficult in aging patients, a rigorous quality control program can ensure that reliable data are obtained in the majority of patients.

Ethnic Differences in Pulmonary Function in Healthy Nonsmoking Asian-Americans and European-Americans—Korotzer B, Ong S, Hansen JE. Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1101-1108.

We investigated ethnic differences in spirometry and gas transfer (DL_{CO}) in a young, healthy population of nonsmoking physicians and medical students aged 22-33 yr, of European or Asian descent. Each answered questions detailing ethnic background, medical history, level of physical activity, and length of residence in the United States. Spirometry and single-breath DL_{CO} maneuvers were performed in accordance with ATS standards. Venous blood was measured for hemoglobin (Hb). The same equipment was used to test all subjects. Data were analyzed by multiple linear regression. Eighty subjects were studied, with 20 in each of the following groups: European male, European female, Asian male, and



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Asian female. Asian values for forced vital capacity, forced expiratory volume in 1 s (FEV_1), and alveolar volume (V_A) were significantly lower than for Europeans, but DL_{CO}, DL_{CO}/V_A, and DL_{CO}/V_A/Hb did not differ significantly. These differences could not be attributed to age, length of residence in the United States, activity level, or variance in baseline characteristics and anthropometric measurements, and therefore represent a true physiologic difference. Ethnic differences between individuals of Asian and European backgrounds should be considered when interpreting pulmonary function tests, especially when predicted values are based on populations of European descent.

Patient Preferences Regarding Possible Outcomes of Lung Resection: What Outcomes Should Preoperative Evaluations Target? Cykert S, Kissling G, Hansen CJ. Chest 2000 Jun;117(6):1551-1559.

CONTEXT: Lung resection can lead to significant postoperative complications: Although many reports describe the likelihood of postoperative problems, such as atelectasis, pneumonia, and prolonged ventilator dependence, it is unclear whether patients perceive these outcomes as sufficiently severe to influence their decisions about surgery. OBJECTIVE: To assess patients' preferences regarding possible outcomes of lung resection, including traditional complications reported in the lung surgery literature and outcomes that describe functional limitation. DESIGN: Utility analysis. SETTING: A community hospital internal medicine clinic, a private internal medicine practice, and a private pulmonary practice. PARTICIPANTS: Sixty-four patients, aged 50 to 75 years, who were awaiting appointments at the designated clinic sites. Main outcome measure: Patients' strength of preference regarding potential outcomes of lung resection as derived from health utility scores. RESULTS: Common postoperative complications were assigned high utility scores by patients. On a scale for which 1.0 represents perfect health and 0 represents death, postoperative atelectasis, pneumonia, and 3 days of mechanical ventilation were all rated >0.75. Scores describing limited physical function were strikingly low. Specifically, activity limited to bed to chair movement and the need for complete assistance with activities of daily living were all assigned utility scores <0.2. Twenty-four-hour oxygen dependence was scored at 0.33. Presence or absence of pulmonary illness did not predict scores for any outcome. CONCLUSIONS: Whether patients suffer from chronic lung disease or not, they do not regard the postoperative outcomes reported in the lung surgery literature as sufficiently morbid to forego important surgery. However, physical debility is perceived as extremely undesirable, and anticipation of its occurrence could deter surgery. Therefore, identification of preoperative predictors of postoperative physical debility would be invaluable for counseling patients who face difficult decisions about lung resection.

Lung Volume Reduction Surgery: A Survey on the European Experience—Hamacher J, Russi EW, Weder W. Chest 2000 Jun;117(6):1560-1567.

STUDY OBJECTIVE: To evaluate the activity and evolution in the field of lung volume reduction surgery (LVRS) performed at surgical centers in Europe. BACKGROUND: LVRS is a novel surgical therapy with the potential to improve lung function, exercise performance, and quality of life in selected patients suffering from severe pulmonary emphysema. METHODS: Questionnaire addressed to 75 European thoracic surgical centers presumed to perform LVRS, and review of the literature. RESULTS: Of 45 responding centers, 42 centers in 17 countries covering a population of 423 million reported performing LVRS. Until the end of 1998, 1,120 patients were reported to have undergone LVRS, corresponding to 2.6 patients/million inhabitants. Thirty-one of 40 centers (78%) perform the operation bilaterally. Most centers (83%) evaluate their activity prospectively. The average perioperative mortality rate of 4.1% is moderate. The most commonly utilized technique is video-assisted thoracoscopy, which is most frequently performed bilaterally. Two thirds of the centers treat patients with $alpha_1$-antitrypsin deficiency, and half of the centers will consider patients with homogenous morphology of emphysema on CT scan for LVRS. Half of the centers also perform lung transplantation. The five largest centers have operated on 49% of all LVRS patients assessed by this survey. CONCLUSIONS: LVRS is performed at few thoracic surgical centers throughout Europe, with a large variation in the operative activity between different regions. Half of the centers also perform lung transplantation. Between 1995 and 1997, the number of LVRS procedures performed per year nearly tripled but has reached a plateau since then. As five centers perform nearly half the total number of operations, an optimal exchange of knowledge with smaller centers seems important.

Sleep Medicine Practices, Training, and Attitudes: A Wake-Up Call for Pulmonologists—Phillips B, Collop N, Goldberg R. Chest 2000 Jun;117(6):1603-1607.

STUDY OBJECTIVES: To determine attitudes and knowledge about sleep medicine among chest physicians. DESIGN: Interactive survey of self-selected respondents. SETTING: Interactive session at the 1998 American College of Chest Physicians (ACCP) annual meeting. PARTICIPANTS: Approximately 60 chest physicians. INTERVENTIONS: Interactive questions about the knowledge, training, attitudes, and practice of sleep medicine. Measurements and results: Response rates demonstrated that 65% of respondents directed or were on the staff of a sleep laboratory, 18% had American Board of Sleep Medicine (ABSM) certification, and only 3% had completed formal sleep medicine training, and performance on test questions about sleep-disordered breathing was better than that on questions about "nonpulmonary" sleep disorders. We polled approximately 60 participants in an interactive session called "Issues in Sleep Medicine Education and Practice" at the ACCP annual meeting in October 1998. The group was well-credentialed, with about one third of

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participants being board-certified in pulmonary medicine and critical care medicine, and about 17% having passed the ABSM examination. About two thirds of the group spent ≤ 25% of their time in the practice of sleep medicine, but > 30% directed sleep laboratories. Respondents thought that sleep training was better addressed in pulmonary fellowship training than in medical school or other postgraduate training experiences. Forty-three percent of the group had received training in sleep medicine as part of a pulmonary fellowship. About half of the sample thought that formal training should be required for eligibility to take the ABSM examination. When presented with two "nonpulmonary" sleep disorder cases, this well-trained and self-selected group did not perform very well. The findings suggest that pulmonologists are actively involved in the practice of sleep medicine and that they both need and desire formal training in sleep disorders during pulmonary fellowship training. CONCLUSIONS: Participants were actively involved in the practice of sleep medicine, most had trained informally, and performance on questions about nonpulmonary sleep disorders was not good.

Nasal Continuous Positive Airway Pressure Use in Children with Obstructive Sleep Apnea Younger than 2 Years of Age—Downey R 3rd, Perkin RM, MacQuarrie J. Chest 2000 Jun;117(6):1608-1612.

STUDY OBJECTIVES: To assess the efficacy of continuous positive airway pressure (CPAP) in obstructive sleep apnea (OSA) patients who are < 2 years of age. DESIGN: A retrospective chart review of 18 patients from 1992 to 1999 who had OSA confirmed by polysomnography. All patients in this study also completed a separate night of CPAP polysomnography to determine the effectiveness of CPAP in the correction of OSA. Nasal CPAP compliance data were gathered via clinical follow-up examination, telephone interview, or mailed questionnaire. SETTING: All patients were studied in the Sleep Disorders Center at Loma Linda University Children's Hospital in Loma Linda, CA. PATIENTS: All patients were < 2 years old. INTERVENTION: After OSA was confirmed by the results of technician-attended nocturnal polysomnography, separate technician-attended nocturnal CPAP polysomnography was completed. On CPAP nights, CPAP pressure was titrated to ameliorate OSA and snoring. CPAP pressure was increased by 2-cm H_2O or 1-cm H_2O increments. RESULTS: Data were analyzed by dependent groups t test at $p < 0.05$ level of significance. CPAP statistically improved respiratory parameters significantly when compared to baseline polysomnography. The following four patient subgroups emerged from the analysis: group 1 consisted of six patients who had tracheostomies prior to the CPAP trial, with two patients using CPAP as an alternative to tracheostomy; group 2 consisted of two patients who had previous unsuccessful adenostonsillectomies and who used CPAP successfully, with both having OSA resolution over time; group 3 consisted of four patients who did not tolerate CPAP on the study night; and group 4 consisted of six patients who used CPAP nightly, had OSA resolution over time, and therefore, no longer needed CPAP therapy. Thus, 10 of 18 patients used CPAP either on an interim basis for corrective therapy or as a primary treatment modality for OSA. CONCLUSIONS: These data show that children < 2 years of age can tolerate and use CPAP effectively. In several cases, CPAP treatment could be discontinued as OSA resolved over time. The reasons for this are discussed in the text.

Evaluation of the Short-Form 36-Item Questionnaire to Measure Health-Related Quality of Life in Patients with Idiopathic Pulmonary Fibrosis—Martinez TY, Pereira CA, dos Santos ML, Ciconelli RM, Guimaraes SM, Martinez JA. Chest 2000 Jun;117(6):1627-1632.

OBJECTIVE: To validate the use of the 36-item short-form questionnaire (SF-36) for measuring health-related quality of life (HRQL) in patients

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with idiopathic pulmonary fibrosis (IPF). DESIGN: Observational data at a single point in time. SETTING: A specialized outpatient respiratory clinic. PARTICIPANTS: Thirty-four patients (mean ± SE age, 58.29 ± 1.87 years) with IPF and no significant comorbidity. A matched control group for HRQL measurements was composed of 34 normal subjects (mean age, 58.00 ± 1.89 years). Measurements and results: Dyspnea was measured by the baseline dyspnea index (BDI). Respiratory function evaluation included FVC, FEV_1, and resting arterial blood gases. IPF patients showed a mean BDI score of 5.21 ± 0.46. The mean FVC and FEV_1 values were 62.41 ± 2.96% and 66.41 ± 3.33%, respectively. The mean P_{aO_2} was 67 ± 2.51 mm Hg, and the mean P_{aCO_2} was 37 ± 1. 05 mm Hg. Patients scored significantly worse than control subjects with respect to the SF-36 domains of physical functioning, physical role, general health perceptions, vitality, social functioning, emotional role, and mental health index. BDI scores were significantly correlated with five SF-36 components, and FVC and FEV_1 were significantly correlated with two SF-36 components. Significant negative correlations were found between arterial pH and four SF-36 domains. CONCLUSIONS: Patients with IPF have a significant impairment of HRQL in both physical and psychological functioning. Dyspnea is the most important factor influencing the quality of life in these subjects. The SF-36 questionnaire is a valid instrument to evaluate HRQL in IPF patients.

International Comparison of Median Age at Death from Cystic Fibrosis—Fogarty A, Hubbard R, Britton J. Chest 2000 Jun;117(6):1656-1660.

STUDY OBJECTIVES: To compare international trends in mortality from cystic fibrosis. DESIGN: Comparison of trends in median age at death using national mortality data. SETTING: Data from 10 countries in North America, Europe, and Australasia. PARTICIPANTS: All persons registered as having died of cystic fibrosis in specified years from 1980 to 1994. INTERVENTIONS: Comparison of relative odds of death at the international median age at death for the year of death between countries for two periods of time; from 1980 to 1987 (10 countries) and from 1980 to 1994 (7 countries). Measurements and results: The international median age at death increased from 8 years in 1974 to 21 years in 1994. Median age at death also increased within all countries, was consistently highest in the United States, and varied significantly by a factor of > twofold between countries. Women were significantly more likely to die at a younger age than the median age at death than men. CONCLUSIONS: Median age at death from cystic fibrosis is increasing, but our findings imply that clinically significant differences in survival with cystic fibrosis persist between countries.

Prospective Randomized Trial Comparing Pressure-Controlled Ventilation and Volume-Controlled Ventilation in ARDS—Esteban A, Alia I, Gordo F, de Pablo R, Suarez J, Gonzalez G, Blanco J. Chest 2000 Jun;117(6):1690-1696.

STUDY OBJECTIVES: To compare in-hospital mortality of patients with ARDS ventilated with either pressure-controlled ventilation (PCV) or volume-controlled ventilation (VCV) with a square-wave inspiratory flow. DESIGN: Multicenter and randomized trial. SETTING: Twelve medical-surgical ICUs located in tertiary-care hospitals. PATIENTS: Seventy-nine patients having ARDS, as defined by the American-European Consensus Conference. INTERVENTIONS: Patients were randomly assigned to be ventilated with either PCV (n = 37) or VCV (n = 42). In both instances, inspiratory plateau pressure was limited to ≤ 35 cm H_2O. MEASUREMENTS AND RESULTS: There were no significant differences among the studied groups at the moment of randomization, although there was a trend toward greater renal failure in patients assigned to VCV. Ventilatory settings and blood gases did not significantly differ over time between the two groups. Patients in the VCV group had both a significantly higher in-hospital mortality rate than those in the PCV group (78% vs 51%, respectively) and a higher number of extrapulmonary organ failures (median, 4 vs 2, respectively). The development of renal failure during the study period was also significantly more frequent among VCV patients (64% vs 32%, respectively). Multivariate analysis showed that factors independently associated with an increased mortality rate were the presence of two or more extrapulmonary organ failures (odds ratio [OR], 4.61; 95% confidence interval [CI], 1.38 to 15.40) and acute renal failure (OR, 3.96; 95% CI, 1.10 to 14.28) but not the ventilatory mode used. CONCLUSIONS: The increased number of extrapulmonary organ failures developed in patients of the VCV group was strongly associated with a higher mortality rate. The development of organ failures was probably not related to the ventilatory mode.

Single-Breath Measurements of Pulmonary Oxygen Uptake and Gas Flow Rates for Ventilator Management in ARDS—Szalados JE, Noe FE, Busby MG, Boysen PG. Chest 2000 Jun;117(6):1805-1809.

Monitoring data in critical care and anesthesiology should be displayed to present a rapid and easily comprehensible definition of the patient's clinical status. A graphic computer display of the analog output of gas flow rates and the O_2 and CO_2 concentrations of respiratory gases profiles the expired breath for an estimation of pulmonary function and gas exchange. An estimate of pulmonary perfusion, cardiac output, and the general adequacy of cardiovascular circulation is obtained from the computer calculation of O_2 uptake and CO_2 elimination, dead space, and alveolar ventilation. Adjunctive data from the spirometric measurements of airway pressures, volumes, and compliance, supplemented by hemodynamic monitoring, aids in the diagnosis of physiologic changes. For > 10 years, we have used this system to monitor patients who are anesthetized, sedated, and receiving mechanical ventilation during anesthesia and surgery, and recently have extended the technique to intensive care areas. Our experience has shown good correlation of changes in the

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computer-assisted expired breath analysis with coinciding clinical events, including upper airway obstruction, bronchospasm, and alveolar volume/pulmonary capillary blood flow impairment. To demonstrate the use of this system, we describe the ventilator management for a patient with severe ARDS. In this patient, changes in ventilator management, including pressure control ventilation, improved pulmonary O_2 uptake (mean, 18.7 vs 8.5 mL/breath), CO_2 elimination (mean, 17 vs 13 mL/breath), and compliance (mean, 29.7 vs 19.0 mL/cm H_2O), were compared with intermittent mandatory ventilation.

Persistent Pneumomediastinum in Interstitial Fibrosis Associated with Rheumatoid Arthritis: Treatment with High-Concentration Oxygen—Patel A, Kesler B, Wise RA. Chest 2000 Jun;117(6):1809-1813.

We present a case of persistent spontaneous pneumomediastinum precipitated by an upper respiratory infection in a patient with interstitial fibrosis associated with rheumatoid arthritis who was receiving chronic corticosteroid treatment. The persistent nature of the mediastinal emphysema over 2 months eventually required treatment with high concentrations of inhaled oxygen that resulted in rapid resolution of the pneumomediastinum without recurrence over 6 months of follow-up. This case, along with others in the medical literature, emphasizes the need for early use of high-concentration inhaled oxygen in the treatment of pneumomediastinum in high-risk patients, such as those with connective tissue disorders.

Relationship of Sputum Color to Nature and Outpatient Management of Acute Exacerbations of COPD—Stockley RA, O'Brien C, Pye A, Hill SL. Chest 2000 Jun;117(6):1638-1645.

Study objectives: To stratify COPD patients presenting with an acute exacerbation on the basis of sputum color and to relate this to the isolation and viable numbers of bacteria recovered on culture. DESIGN: Open, longitudinal study of sputum characteristics and acute-phase proteins. SETTING: Patients presenting to primary-care physicians in the United Kingdom. Patients were followed up as outpatients in specialist clinic. PATIENTS: One hundred twenty-one patients with acute exacerbations of COPD were assessed together with a single sputum sample on the day of presentation (89 of whom produced a satisfactory sputum sample for analysis). One hundred nine patients were assessed 2 months later when they had returned to their stable clinical state. INTERVENTIONS: The expectoration of green, purulent sputum was taken as the primary indication for antibiotic therapy, whereas white or clear sputum was not considered representative of a bacterial episode and the need for antibiotic therapy. RESULTS: A positive bacterial culture was obtained from 84% of patients sputum if it was purulent on presentation compared with only 38% if it was mucoid ($p < 0.0001$). When restudied in the stable clinical state, the incidence of a positive bacterial culture was similar for both groups (38% and 41%, respectively). C-reactive protein concentrations were significantly raised ($p < 0.0001$) if the sputum was purulent (median, 4.5 mg/L; interquartile range [IQR], 6. 2 to 35.8). In the stable clinical state, sputum color improved significantly in the group who presented with purulent sputum from a median color number of 4.0 (IQR, 4.0 to 5.0) to 3.0 (IQR, 2.0 to 4. 0; $p < 0.0001$), and this was associated with a fall in median C-reactive protein level to 2.7 mg/L (IQR, 1.0 to 6.6; $p < 0.0001$). CONCLUSIONS: The presence of green (purulent) sputum was 94.4% sensitive and 77.0% specific for the yield of a high bacterial load and indicates a clear subset of patient episodes identified at presentation that is likely to benefit most from antibiotic therapy. All patients who produced white (mucoid) sputum during the acute exacerbation improved without antibiotic therapy, and sputum characteristics remained the same even when the patients had returned to their stable clinical state.

A Three-Dimensional Model of the Human Pulmonary Acinus—Kitaoka H, Tamura S, Takaki R. J Appl Physiol 2000 Jun;88(6):2260-2268.

A three-dimensional (3-D) model of the human pulmonary acinus, a gas exchange unit, is constructed with a labyrinthine algorithm generating branching ducts that fill a given space completely. Branching down to the third respiratory bronchioles is generated with the proposed algorithm. A subacinus, a region supplied by the last respiratory bronchiole, is approximated to be a set of cubic cells with a side dimension of 0.5 mm. The labyrinthine algorithm is used to determine a pathway through all cells only once, except at branching points with the smallest path lengths. In choosing each step of a pathway, random variables are used. Resulting labyrinths have equal mean path lengths and equal surface areas of inner walls. An alveolus can be generated by attaching alveolar septa, 0.25 mm long and 0.1 mm wide, to the inner walls. Total alveolar surface area and numbers of alveolar ducts, alveolar sacs, and alveoli in our 3-D acinar model are in good accordance with those reported in the literature.

Developmental Aspects of Oxygen Sensing by the Carotid Body—Donnelly DF. J Appl Physiol 2000 Jun;88(6):2296-2301.

The carotid body chemoreceptors, the major hypoxia sensory organs for the respiratory system, undergo a significant increase in their hypoxia responsiveness in the postnatal period. This is manifest by a higher level of afferent nerve activity for a given level of arterial oxygen tension. The mechanism for the enhanced sensitivity is unresolved, but most work has focused on the glomus cell, a secretory cell apposed to the afferent nerve ending and believed to be the site of hypoxia transduction. The glomus cell secretory response to hypoxia increases postnatally, and this is correlated with an enhanced calcium rise in response to hypoxia and an increase in oxygen-sensitive potassium currents. These changes are sensitive to the level of hypoxia in the postnatal period, and significant impairment of organ function is observed with postnatal hypoxia as well as postnatal hyperoxia. Although many questions remain, especially with regard to the coupling of glomus cells to nerve endings, the use of cellular and molecular techniques should offer resolution in the near future.

Oxygen Sensing by the Carotid Body Chemoreceptors—Prabhakar NR. J Appl Physiol 2000 Jun;88(6):2287-2295.

Carotid bodies are sensory organs that detect changes in arterial blood oxygen, and the ensuing reflexes are critical for maintaining homeostasis during hypoxemia. During the past decade, tremendous progress has been made toward understanding the cellular mechanisms underlying oxygen sensing at the carotid body. The purpose of this minireview is to highlight some recent concepts on sensory transduction and transmission at the carotid body. A bulk of evidence suggests that glomus (type I) cells are the initial site of transduction and that they release transmitters in response to hypoxia, which causes depolarization of nearby afferent nerve endings, leading to an increase in sensory discharge. There are two main hypotheses to explain the transduction process that triggers transmitter release. One hypothesis assumes that a biochemical event associated with a heme protein triggers the transduction cascade. The other hypothesis suggests that a K^+ channel protein is the oxygen sensor and that inhibition of this channel by hypoxia leading to depolarization is a seminal event in transduction. Although there is body of evidence supporting and questioning each of these, this review will try to point out that the truth lies somewhere in an interrelation between the two. Several transmitters have been identified in glomus cells, and they are released in response to hypoxia. However, their precise roles in sensory transmission remain uncertain. It is hoped that future studies involving transgenic animals with targeted disruption of genes encoding transmitters and their receptors may resolve some of the key issues surrounding the sensory trans-

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mission at the carotid body. Further studies are necessary to identify whether a single sensor or multiple oxygen sensors are needed for the transduction process.

Mechanisms for Isolated Volume Response to a Bronchodilator in Patients with COPD—Cerveri I, Pellegrino R, Dore R, Corsico A, Fulgoni P, van De Woestijne KP, Brusasco V. J Appl Physiol 2000 Jun;88(6):1989-1995.

We hypothesized that an altered effect of lung inflation on airway caliber may in part explain the isolated volume response to bronchodilators, i.e., an increase of forced vital capacity (FVC) without change in 1-s forced expiratory volume (FEV_1). Small-airway caliber was measured by high-resolution computed tomography at functional residual capacity and total lung capacity in five chronic obstructive pulmonary disease patients with an isolated increase of FVC (FVC responders) and five with an increase of both FVC and FEV_1 (FVC-FEV_1 responders) after inhalation of salbutamol. In FVC-FEV_1 responders, the airway diameter increased with the cube root of increase in lung volume but was unchanged or even decreased in four of five FVC responders. FVC responders had more severe emphysema, as inferred from lung function and imaging studies, than FVC-FEV_1 responders. We speculate that longitudinal traction or space competition (Verbeken EK, Cauberghs M, and Van de Woestijne KP. J Appl Physiol 82468-2480, 1996) are possible underlying mechanisms. We conclude that the isolated volume response to bronchodilators is associated with severe emphysema and likely results from an altered effect of lung inflation on airway caliber.

Analysis of the Mechanisms of Expiratory Asynchrony in Pressure Support Ventilation: A Mathematical Approach—Yamada Y, Du HL. J Appl Physiol 2000 Jun;88(6):2143-2150.

A mathematical model was developed to analyze the mechanisms of expiratory asynchrony during pressure support ventilation (PSV). Solving the model revealed several results. 1) Ratio of the flow at the end of patient neural inspiration to peak inspiratory flow (VTI/V_{peak}) during PSV is determined by the ratio of time constant of the respiratory system (tau) to patient neural inspiratory time (T_I) and the ratio of the set pressure support (Pps) level to maximal inspiratory muscle pressure (Pmus max). 2) VTI/V_{peak} is affected more by tau/T_I than by Pps/Pmus max. VTI/V_{peak} increases in a sigmoidal relationship to tau/T_I. An increase in Pps/Pmus max slightly shifts the VTI/V_{peak}-tau/T_I curve to the right, i.e., VTI/V_{peak} becomes lower as Pps/Pmus max increases at the same tau/T_I. 3) Under the selected adult respiratory mechanics, VTI/V_{peak} ranges from 1 to 85% and has an excellent linear correlation with tau/T_I. 4) In mechanical ventilators, single fixed levels of the flow termination criterion will always have chances of both synchronized termination and asynchronized termination, depending on patient mechanics. An increase in tau/TI causes more delayed and less premature termination opportunities. An increase in Pps/Pmus max narrows the synchronized zone, making inspiratory termination predisposed to be in asynchrony. Increasing the expiratory trigger sensitivity of a ventilator shifts the synchronized zone to the right, causing less delayed and more premature termination. Automation of expiratory trigger sensitivity in future mechanical ventilators may also be possible. In conclusion, our model provides a useful tool to analyze the mechanisms of expiratory asynchrony in PSV.

Causes and Outcomes of the Acute Chest Syndrome in Sickle Cell Disease. National Acute Chest Syndrome Study Group—Vichinsky EP, Neumayr LD, Earles AN, Williams R, Lennette ET, Dean D, et al. N Engl J Med 2000 Jun 22;342(25):1855-1865.

BACKGROUND: The acute chest syndrome is the leading cause of death among patients with sickle cell disease. Since its cause is largely unknown, therapy is supportive. Pilot studies with improved diagnostic techniques suggest that infection and fat embolism are underdiagnosed in patients with the syndrome. METHODS: In a 30-center study, we analyzed 671 episodes of the acute chest syndrome in 538 patients with sickle cell disease to determine the cause, outcome, and response to therapy. We evaluated a treatment protocol that included matched transfusions, bronchodilators, and bronchoscopy. Samples of blood and respiratory tract secretions were sent to central laboratories for antibody testing, culture, DNA testing, and histopathological analyses. RESULTS: Nearly half the patients were initially admitted for another reason, mainly pain. When the acute chest syndrome was diagnosed, patients had hypoxia, decreasing hemoglobin values, and progressive multilobar pneumonia. The mean length of hospitalization was 10.5 days. Thirteen percent of patients required mechanical ventilation, and 3 percent died. Patients who were 20 or more years of age had a more severe course than those who were younger. Neurologic events occurred in 11 percent of patients, among whom 46 percent had respiratory failure. Treatment with phenotypically matched transfusions improved oxygenation, with a 1 percent rate of alloimmunization. One fifth of the patients who were treated with bronchodilators had clinical improvement. Eighty-one percent of patients who required mechanical ventilation recovered. A specific cause of the acute chest syndrome was identified in 38 percent of all episodes and 70 percent of episodes with complete data. Among the specific causes were pulmonary fat embolism and 27 different infectious pathogens. Eighteen patients died, and the most common causes of death were pulmonary emboli and infectious bronchopneumonia. Infection was a contributing factor in 56 percent of the deaths. CONCLUSIONS: Among patients with sickle cell disease, the acute chest syndrome is commonly precipitated by fat embolism and infection, especially community-acquired pneumonia. Among older patients and those with neurologic symptoms, the syndrome often progresses to respiratory failure. Treatment with transfusions and bronchodilators improves oxygenation, and with aggressive treatment, most patients who have respiratory failure recover.

Long-Term Treatment of Primary Pulmonary Hypertension with Aerosolized Iloprost, a Prostacyclin Analogue—Hoeper MM, Schwarze M, Ehlerding S, Adler-Schuermeyer A, Spiekerkoetter E, Niedermeyer J, et al. N Engl J Med 2000 Jun 22;342(25):1866-1870.

BACKGROUND: Continuous intravenous infusion of epoprostenol (prostacyclin) is an effective treatment for primary pulmonary hypertension. This approach requires the insertion of a permanent central venous catheter, with the associated risk of serious complications. Recently, aerosolized iloprost, a stable prostacyclin analogue, has been introduced as an alternative therapy for severe pulmonary hypertension. METHODS: We evaluated the effects of aerosolized iloprost on exercise capacity and hemodynamic variables over a one-year period in patients with primary pulmonary hypertension. RESULTS: Twenty-four patients with primary pulmonary hypertension received aerosolized iloprost at a daily dose of 100 or 150 microg for at least one year. The mean (±SD) distance covered in the six-minute walk test increased from 278±96 m at base line to 363±135 m after 12 months ($p< 0.001$). During the same period, the mean pulmonary arterial pressure before the inhalation of iloprost declined from 59±10 mm Hg to 52±15 mm Hg ($p=0.006$), cardiac output increased from 3.8±1.4 liters per minute to 4.4±1.3 liters per minute ($p=0.02$), and pulmonary vascular resistance declined from 1205±467 dyn × sec × cm^{-5} to 925±469 dyn × sec × cm^{-5} ($p<0.001$). The treatment was generally well tolerated, except for mild coughing, minor headache, and jaw pain in some patients. CONCLUSIONS: Long-term treatment with aerosolized iloprost is safe and has sustained effects on exercise capacity and pulmonary hemodynamics in patients with primary pulmonary hypertension.

Cerebral Oxygenation during Hemorrhagic Shock: Perils of Hyperventilation and the Therapeutic Potential of Hypoventilation—Man-

ley GT, Hemphill JC, Morabito D, Derugin N, Erickson V, Pitts LH, Knudson MM. J Trauma 2000 Jun;48(6):1025-1032; discussion 1032-1033.

OBJECTIVES: Prophylactic hyperventilation of patients with head injuries worsens outcome, presumably by exacerbating tissue hypoxia. Oxygen tension in brain tissue ($PbrO_2$) provides a direct measurement of cerebral metabolic substrate delivery and varies with changing end-tidal carbon dioxide tension ($ETCO_2$) and mean arterial pressure. However, the effects of hyperventilation and hypoventilation on $PbrO_2$ during hemorrhagic shock are not known. The aim of this study was to examine the effects of alteration in ventilation on $PbrO_2$ in hemorrhaged swine. METHODS: Clark-type polarographic probes were inserted into the brain tissue of seven swine to measure $PbrO_2$ directly. To examine the effects of alterations in ventilation on hemorrhage-induced hypotension, swine were hemorrhaged to 50% estimated blood volume and $PbrO_2$ was monitored during hyperventilation (RR = 30) and hypoventilation (RR = 4). RESULTS: After the 50% hemorrhage, $PbrO_2$ declined rapidly from 39.8 ± 4.6 mm Hg to 11.4 ± 2.2 mm Hg. Hyperventilation resulted in a further 56% mean decrease in $PbrO_2$. Hypoventilation produced a 166% mean increase in $PbrO_2$. These changes were significant ($p = 0.001$) for absolute and percentage differences from baseline. CONCLUSION: During hemorrhage, alterations in ventilation significantly changed $PbrO_2$: hyperventilation increased brain-tissue hypoxia whereas hypoventilation alleviated it. This finding suggests that hyperventilation has deleterious effects on brain oxygenation in patients with hemorrhagic shock and those with head trauma. Conversely, hypoventilation with resultant hypercapnia may actually help resolve hemorrhagic shock-induced cerebral hypoxia.

Rib Fractures in the Elderly—Bulger EM, Arneson MA, Mock CN, Jurkovich GJ. J Trauma 2000 Jun;48(6):1040-1046; discussion 1046-1047.

BACKGROUND: We sought to ascertain the extent to which advanced age influences the morbidity and mortality after rib fractures (fxs), to define the relationship between number of rib fractures and morbidity and mortality, and to evaluate the influence of analgesic technique on outcome. METHODS: A retrospective cohort study involving all 277 patients ≥ 65 years old with rib fxs admitted to a Level I trauma center over 10 years was undertaken. The control group consisted of 187 randomly selected patients, 18 to 64 years old, with rib fxs admitted over the same time period. Outcomes included pulmonary complications, number of ventilator days, length of intensive care unit and hospital stay (LOS), disposition, and mortality. The specific analgesic technique used was also examined. RESULTS: The two groups had similar mean number of rib fxs (3.6 elderly vs. 4.0 young), mean chest Abbreviated Injury Scores (3.0 vs. 3.0), and mean Injury Severity Score (20.7 vs. 21.4). However, mean number of ventilator days (4.3 vs. 3.1), intensive care unit days (6.1 vs. 4.0), and LOS (15.4 vs. 10.7 days) were longer for the elderly patients. Pneumonia occurred in 31% of elderly versus 17% of young ($p < 0.01$) and mortality was 22% for the elderly versus 10% for the young ($p < 0.01$). Mortality and pneumonia rates increased as the number of rib fxs increased with and odds ratio for death of 1.19 and for pneumonia of 1.16 per each additional rib fracture ($p < 0.001$). The use of epidural analgesia in the elderly (LOS >2 days) was associated with a 10% mortality versus 16% without the use of an epidural ($p = 0.28$). In the younger group (LOS >2 days), mortality with and without the use of an epidural was 0% and 5%, respectively. CONCLUSION: Elderly patients who sustain blunt chest trauma with rib fxs have twice the mortality and thoracic morbidity of younger patients with similar injuries. For each

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additional rib fracture in the elderly, mortality increases by 19% and the risk of pneumonia by 27%. As the number of rib fractures increases, there is a significant increase in morbidity and mortality in both groups, but with different patterns for each group. Further prospective study is needed to determine the utility of epidural analgesia in this population.

Asthma Aggravation, Combustion, and Stagnant Air—Norris G, Larson T, Koenig J, Claiborn C, Sheppard L, Finn D. Thorax 2000 Jun; 55(6):466-470.

BACKGROUND: The relationship between current concentrations of ambient air pollution and adverse health effects is controversial. We report a meteorological index of air stagnation that is associated with daily visits to the emergency department for asthma in two urban areas. METHODS: Data on daily values of a stagnation persistence index and visits to the emergency department for asthma were collected for approximately two years in Spokane, Washington, USA and for 15 months in Seattle, Washington, USA. The stagnation persistence index represents the number of hours during the 24 hour day when surface wind speeds are less than the annual hourly median value, an index readily available for most urban areas. Associations between the daily stagnation persistence index and daily emergency department visits for asthma were tested using a generalised additive Poisson regression model. A factor analysis of particulate matter ($PM_{2.5}$) composition was performed to identify the pollutants associated with increased asthma visits. RESULTS: The relative rate of the association between a visit to the emergency department for asthma and the stagnation persistence index was 1.12 (95% CI 1.05 to 1.19) in Spokane and 1.21 (95% CI 1.09 to 1.35) in Seattle for an increase of 11 and 10 hours, respectively, of low wind speed in a given day. The stagnation persistence index was only correlated with one set of factor loadings; that cluster included the stagnation persistence index, carbon monoxide, and organic/elemental carbon. CONCLUSION: Increased air stagnation was shown to be a surrogate for accumulation of the products of incomplete combustion, including carbon monoxide and fine particulate levels of organic and elemental carbon, and was more strongly associated with asthma aggravation than any one of the measured pollutants.

Asthma after Childhood Pneumonia: Six Year Follow Up Study—Clark CE, Coote JM, Silver DA, Halpin DM. BMJ 2000 Jun 3;320(7248): 1514-1516.

OBJECTIVE: To establish the long-term cumulative prevalence of asthma in children admitted to hospital with pneumonia and to examine the hypothesis that some children admitted to hospital with pneumonia may be presenting with undiagnosed asthma. DESIGN: Prospective study of a cohort of children previously admitted to hospital with pneumonia, followed up by postal questionnaires to their general practitioners and the children or their parents. SETTING: General practices in southwest England. PARTICIPANTS: 78 children admitted to the Royal Devon and Exeter Hospital between 1989 and 1991 with a diagnosis of pneumonia confirmed on independent review of x-ray films. MAIN OUTCOME MEASURES: Any diagnosis of asthma, use of any treatment for asthma, and asthma symptom scores. RESULTS: On the basis of a 100% response rate from general practitioners and 86% from patients or parents, the cumulative prevalence of asthma was 45%. A diagnosis of asthma was associated with a family history of asthma (odds ratio 11.23; 95% confidence interval 2.57 to 56.36; p=0.0002). Mean symptom scores were higher for all children with asthma (mean score 2.4; chi^2=14.88; p=0. 0001) and for children with asthma not being treated (mean 1.4; chi^2=6.2; p=0.01) than for those without asthma (mean 0.2). CONCLUSIONS: A considerable proportion of children presenting to a district general hospital with pneumonia either already have unrecognised asthma or subsequently develop asthma. The high cumulative prevalence of asthma suggests that careful follow up of such children is worth while. Asthma is undertreated in these children; a structured symptom questionnaire may help to identify and reduce morbidity due to undertreatment.

Systematic Review of Studies of Patient Satisfaction with Telemedicine—Mair F, Whitten P. BMJ 2000 Jun 3;320(7248):1517-1520.

OBJECTIVE: To review research into patient satisfaction with teleconsultation, specifically clinical consultations between healthcare providers and patients involving real time interactive video. DESIGN: Systematic review of telemedicine satisfaction studies. Electronic databases searched include MEDLINE, Embase, Science Citation Index, Social Sciences Citation Index, Arts and Humanities Citation Index, and the TIE (Telemedicine Information Exchange) database. Subjects: Studies conducted worldwide and published between 1966 and 1998. MAIN OUTCOME MEASURES: Quality of evidence about patient satisfaction. RESULTS: 32 studies were identified. Study methods used were simple survey instruments (26 studies), exact methods not specified (5), and qualitative methods (1). Study designs were randomised controlled trial (1 trial); random patient selection (2); case-control (1); and selection criteria not specified or participants represented consecutive referrals, convenience samples, or volunteers (28). Sample sizes were $\leq$ 20 (10 trials), $\leq$ 100 (14), >100 (7), and not specified (1). All studies reported good levels of patient satisfaction. Qualitative analysis revealed methodological problems with all the published work. Even so, important issues were highlighted that merit further investigation. There is a paucity of data examining patients' perceptions or the effects of this mode of healthcare delivery on the interaction between providers and clients. CONCLUSIONS: Methodological deficiencies (low sample sizes, context, and study designs) of the published research limit the generalisability of the findings. The studies suggest that teleconsultation is acceptable to patients in a variety of circumstances, but issues relating to patient satisfaction require further exploration from the perspective of both clients and providers.

Surgical Treatment of Pulmonary Aspergilloma: Current Outcome—Babatasi G, Massetti M, Chapelier A, Fadel E, Macchiarini P, Khayat A, Dartevelle P. J Thorac Cardiovasc Surg 2000 May;119(5):906-912.

OBJECTIVE: This retrospective study was designed to confirm that aggressive pulmonary resection can provide effective long-term palliation of disease for patients with pulmonary aspergilloma. METHODS AND RESULTS: From 1959 to 1998, 84 patients underwent a total of 90 operations for treatment of pulmonary aspergilloma in the Marie-Lannelongue Hospital. The mean follow-up period was 9 years, and 83% of the patients were followed up for 5 years or until death, if the latter occurred earlier. The median age was 44 years. The most common indications were hemoptysis (66%) and sputum production (15%). Fifteen patients (18%) had no symptoms. Tuberculosis and lung abscess were the most common underlying causes of lung disease (65%). The procedures were 70 lobar or segmental resections, 8 cavernostomies, and 7 pneumonectomies. Five thoracoplasties were required after lobectomy (3 patients) or pneumonectomy (2 patients). The operative mortality rate was 4%. The major complications were bleeding (23 patients), prolonged air leak (31 patients), respiratory failure (10 patients), and empyema (5 patients). The actuarial survival curve showed 84% survival at 5 years and 74% survival at 10 years. During the first 2 years, death was related to the surgical procedure and the underlying disease. In contrast, 85% of the survivors had a good late result. CONCLUSION: Lobar resection in both the symptomatic and the asymptomatic patients was conducted in low-risk settings. For patients whose condition is unfit for pulmonary resection, cavernostomy may need to be undertaken despite the high operative risk. The better survival rate in this study may have been due to the selection of patients with better lung function and localized pulmonary disease.

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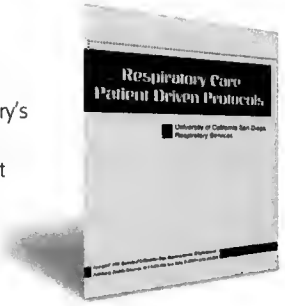
CLINICAL PRACTICE GUIDELINES (CPGS)/PROTOCOLS

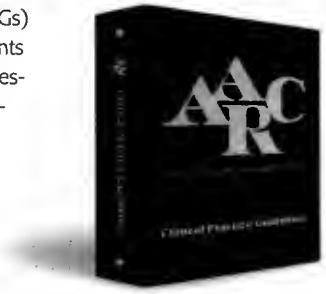


Respiratory Care Patient-Driven Protocols

Written by UCSD faculty, one of the country's leading institutions in the development of protocols. This book serves as an excellent resource in the processes of developing, implementing, or refining care plans. Features 25 complete protocols including: Oxygen, Oximetry, MDI, SVN, PEP, Thoracoscopy, IPPB, Extubation, Secretion Management, and others. Also includes a valuable introduction on how to implement PDPs in your facility. Authors include Jan Phillips-Clar, BS, RRT; Richard Ford, BS, RRT; Timothy Morris, MD; and David Burns, MD. Softcover. 205 pages. Published in 1998.

Item PA801 $85.00 ($99.00 Nonmembers)

Clinical Practice Guidelines



AARC Clinical Practice Guidelines (CPGs) are systematically developed statements to help clinicians deliver appropriate respiratory care in specific clinical circumstances. Individual CPGs include: Spirometry, 1996 Update; Oxygen Therapy in the; Acute Care Hospital ; Nasotracheal Suctioning ; Patient-Ventilator System Checks ; Directed Cough; In-Vitro pH and Blood Gas Analysis and Hemoximetry; Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy; Sampling for Arterial Blood Gas Analysis; Endotracheal Suctioning of Mechanically Ventilated Adults and Children with Artificial Airways; Incentive Spirometry; Postural Drainage Therapy; Bronchial Provocation; Selection of Aerosol Delivery Device; Pulse Oximetry; Single-Breath Carbon Monoxide Diffusing Capacity; Oxygen Therapy in the Home or Extended Care Facility; Exercise Testing for Evaluation of Hypoxemia and/or Desaturation; Humidification during Mechanical Ventilation; Transport of the Mechanically Ventilated Patient; Resuscitation in Acute Care Hospitals; Bland Aerosol Administration; Fiberoptic Bronchoscopy Assisting; Intermittent Positive Pressure Breathing; Application of CPAP to Neonates via Nasal Prongs or Nasopharyngeal Tube; Delivery of Aerosols to the Upper Airway; Neonatal Time-Triggered, Pressure-Limited, Time-Cycled Mechanical Ventilation; Static Lung Volumes; Surfactant Replacement Therapy; Ventilator Circuit Changes ; Metabolic Measurement Using Indirect Calorimetry during Mechanical Ventilation; Transcutaneous Blood Gas Monitoring for Neonatal & Pediatric Patients; Body Plethysmography; Capillary Blood Gas Sampling for Neonatal & Pediatric Patients; Defibrillation during Resuscitation; Infant/Toddler Pulmonary Function Tests; Management of Airway Emergencies; Assessing Response to Bronchodilator Therapy at Point of Care; Discharge Planning for the Respiratory Care Patient; Long-Term Invasive Mechanical Ventilation in the Home; Capnography/Capnometry during Mechanical Ventilation; Selection of an Aerosol Delivery Device for Neonatal and Pediatric Patients; Polysomnography; Selection of an Oxygen Delivery Device for Neonatal and Pediatric Patients; Selection of a Device for Delivery of Aerosol to the Lung Parenchyma; Training the Health-Care Professional for the Role of Patient and Caregiver Educator; Providing Patient and Caregiver Training; Removal of the Endotracheal Tube; Suctioning of the Patient in the Home; Selection of Device, Administration of Bronchodilator, and Evaluation of Response to Therapy in Mechanically Ventilated Patients; Three-ring, loose-leaf binder includes 46 CPGs.

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Discusses therapist-driven protocol (TDP) implementation strategies, research, staff selection, training, obstacles, attitudes of other staff, and the results of TDP implementation. Featuring James K. Stoller, MD, and Sam P. Giordano, MBA, RRT. 90-min. videotape.

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Therapist-Driven Protocols in Respiratory Care

Learn how therapist-driven protocols can assist practitioners in providing better patient care and containing costs. Provides an overview to the challenges of implementing protocols and discusses how to gain the support of key players on the health care team. Featuring George G. Burton, MD, and Sam P. Giordano, MBA, RRT. 90-min. videotape.

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This simulation involves an 18-year-old man entering the ER intoxicated, with a possible drug overdose. The user performs initial assessment that includes ventilatory parameters. During the course of the simulation, the patient is transferred to the ICU for further monitoring, then deteriorates and requires oxygen therapy via a |40 percent T-piece and mechanical ventilator. The patient is gradually weaned and extubated with supplemental oxygen therapy. CAI Software. Requires Windows 3.1 or higher.

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Emergency Respiratory Care: The Respiratory Care Practitioner's Role

Reviews new approaches to emergency care, "tricks of the trade" in airway maintenance, and changes in the ACLS philosophy and treatment protocols. Discusses how to increase participation in the hospital emergency response team and the obstacles the team must overcome. Featuring Charles G. Durbin, Jr., MD, FCCM, and Richard D. Branson, BS, RRT. 90-min. videotape.

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Epiglottitis Simulation

Presents a 4-year-old girl entering the ER with a "cold" and elevated temperature. A lateral neck x-ray reveals massive edema of the epiglottis, whereupon the child is taken to surgery and intubated. The user is asked to recommend appropriate airway and size for the patient. After further assessment, CPAP is initiated with room air, and two days later the patient is evaluated for extubation and postextubation therapy. CAI Software. Requires Windows 3.1 or higher. 31/2" floppy PC disk.

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Head Trauma Simulation

Simulates the initial evaluation and recommendations for care of a 25-year-old involved in a motor vehicle accident. After evaluation in the ER, the patient is taken to the OR for a craniotomy. Assessment following surgery (including measurement of ICP) indicates the need for CMV, whereupon the user is required to evaluate the effect of CMV, hyperinflation, and ET suctioning on ICP and take appropriate steps to maintain a normal ICP. CAI Software. Requires Windows 3.1 or higher. 31/2" floppy PC disk.

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Teaches identification of the five physical signs indicative of head trauma and explains the development of respiratory failure secondary to trauma. Also discusses airway management, drug therapy, ventilator parameters, acid-base status, and measures to be taken to maintain the appropriate pH, PaCO2 and PaO2. *Individual Independent Study Package.* Softcover book.

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Involves the initial assessment of a 35-year-old man with a history of allergic asthma. Low-flow oxygen is administered initially, followed by bronchodilator therapy. As the simulation progresses, the patient is intubated and receives mechanical ventilation. The user is required to make initial settings and adjustments according to ABG results and patient response. Also included in the simulation is a switch to IMV mode, sedation, and eventual extubation and placement on a 40 percent aerosol mask. CAI Software. Requires Windows 3.1 or higher. 31/2" floppy PC disk.

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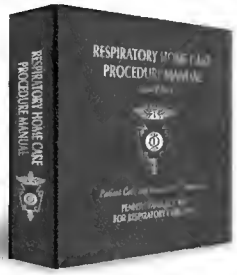
HOME CARE

Respiratory Home Care Procedure Manual



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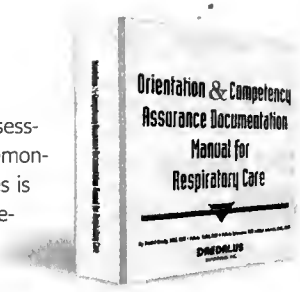

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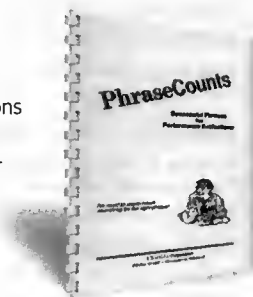

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The Incidence and Pathogenesis of Cardiopulmonary Deterioration after Abrupt Withdrawal of Inhaled Nitric Oxide—Christenson J, Lavoie A, O'Connor M, Bhorade S, Pohlman A, Hall JB Am J Respir Crit Care Med 2000 May;161(5):1443-1449.

We studied the effect of abrupt discontinuation of inhaled nitric oxide (iNO) in patients receiving this drug for treatment of acute hypoxemic respiratory failure (AHRF), in order to determine the need for continued therapy, the incidence and nature of adverse events, and the risk factors predicting these adverse events. Thirty-one patients who showed an initial increase in P_{aO_2} of > 20 mm Hg in response to iNO underwent a discontinuation trial at 10 to 30 h after beginning iNO. Indwelling arterial and pulmonary artery catheters facilitated monitoring of hemodynamic and gas-exchange parameters. For the group, discontinuation of iNO caused a significant decrease in P_{aO_2}, arterial and mixed venous oxygen saturation, and ratio of P_{aO_2} to fraction of inspired oxygen (F_{IO_2}). Three patterns of response were observed. Eight of 31 (25.8%) patients had minimal changes in oxygenation or hemodynamics, suggesting no need for ongoing therapy. Fifteen of 31 (48%) patients had worsened gas exchange as a predominant response. Eight of 31 patients exhibited hemodynamic collapse, defined as > 20% fall in cardiac output and/or mean arterial blood pressure. In this last subgroup, the pattern of cardiovascular changes suggested that this response arose from an acute increase in right ventricular afterload, and was not a consequence of gas-exchange abnormalities. In all cases, reinstitution of iNO promptly reversed worsened hemodynamics and gas exchange. Independent factors associated with an increased risk of cardiovascular collapse included multisystem organ failure, older age, and initial blood pressure increase in response to iNO; a smaller change in the ratio of P_{aO_2} to F_{IO_2} with initiation of iNO therapy also tended to correlate with this phenomenon. We conclude that careful and monitored discontinuation of iNO in patients with AHRF will identify substantial fractions of patients who are either receiving no benefit from this therapy or who require iNO to maintain an adequate circulation and are therefore at risk for adverse outcome with transport or inadvertent discontinuation of iNO. Future trials of iNO should recognize this complication of such therapy and include assessments for it.

How Is Mechanical Ventilation Employed in the Intensive Care Unit? An International Utilization Review—Esteban A, Anzueto A, Alia I, Gordo F, Apezteguia C, Palizas F, et al. Am J Respir Crit Care Med 2000 May;161(5):1450-1458.

A 1-d point-prevalence study was performed with the aim of describing the characteristics of conventional mechanical ventilation in intensive care units (ICUs from North America, South America, Spain, and Portugal. The study involved 412 medical-surgical ICUs and 1,638 patients receiving mechanical ventilation at the moment of the study. The main outcome measures were characterization of the indications for initiation of mechanical ventilation, the artificial airways used to deliver mechanical ventilation, the ventilator modes and settings, and the methods of weaning. The median age of the study patients was 61 yr, and the median duration of mechanical ventilation at the time of the study was 7 d. Common indications for the initiation of mechanical ventilation included acute respiratory failure (66%), acute exacerbation of chronic obstructive pulmonary disease (13%), coma (10%), and neuromuscular disorders (10%). Mechanical ventilation was delivered via an endotracheal tube in 75% of patients, a tracheostomy in 24%, and a facial mask in 1%. Ventilator modes consisted of assist/control ventilation in 47% of patients and 46% were ventilated with synchronized intermittent mandatory ventilation, pressure support, or the combination of both. The median tidal volume setting was 9 mL/kg in patients receiving assist/control and the median setting of pressure support was 18 cm H_2O. Positive end-expiratory pressure was not employed in 31% of patients. Method of weaning varied considerably from country to country, and even within a country several methods were in use. We conclude that the primary indications for mechanical ventilation and the ventilator settings were remarkably similar across countries, but the selection of modes of mechanical ventilation and methods of weaning varied considerably from country to country.

Recruitment Maneuvers in Three Experimental Models of Acute Lung Injury. Effect on Lung Volume and Gas Exchange—Kloot TE, Blanch L, Melynne Youngblood A, Weinert C, Adams AB, Marini JJ, et al. Am J Respir Crit Care Med 2000 May;161(5):1485-1494.

Recruitment maneuvers (RM), consisting of sustained inflations at high airway pressures, have been advocated as an adjunct to mechanical ventilation in acute respiratory distress syndrome (ARDS). We studied the effect of baseline ventilatory strategy and RM on end-expiratory lung volume (EELV) and oxygenation in 18 dogs, using three models of acute lung injury (ALI; n = 6 in each group): saline lavage (LAV), oleic acid injury (OAI), and intratracheal instillation of *Escherichia coli* (pneumonia; PNM). All three models exhibited similar degrees of lung injury. The PNM model was less responsive to positive end-expiratory pressure (PEEP) than was the LAV or OAI model. Only the LAV model showed an oxygenation response to increasing tidal volume (V_T). After RM, there were transient increases in P_{aO_2} and EELV when ventilating with PEEP = 10 cm H_2O. At PEEP = 20 cm H_2O the lungs were probably fully recruited, since the plateau airway pressures were relatively high (approximately 45 cm H_2O) and the oxygenation was similar to preinjury values, thus making the system unresponsive to RM. Sustained improvement in oxygenation after RM was seen in the LAV model when ventilating with PEEP = 10 cm H_2O and V_T = 15 mL/kg. Changes in EELV correlated with changes in P_{aO_2} only in the OAI model with PEEP = 10 cm H_2O. We conclude that responses to PEEP, V_T, and RM differ among these models of ALI. RM may have a role in some patients with ARDS who are ventilated with low PEEP and low V_T.

Implications of Extubation Delay in Brain-Injured Patients Meeting Standard Weaning Criteria—Coplin WM, Pierson DJ, Cooley KD, Newell DW, Rubenfeld GD Am J Respir Crit Care Med 2000 May; 161(5):1530-1536.

We hypothesized that variation in extubating brain injured patients would affect the incidence of nosocomial pneumonia, length of stay, and hospital charges. In a prospective cohort of consecutive, intubated brain-injured patients, we evaluated daily: intubation status, spontaneous ventilatory parameters, gas exchange, neurologic status, and specific outcomes listed above. Of 136 patients, 99 (73%) were extubated within 48 h of meeting defined readiness criteria. The other 37 patients (27%) remained intubated for a median 3 d (range, 2 to 19). Patients with delayed extubation developed more pneumonias (38 versus 21%, $p < 0.05$) and had longer intensive care unit (median, 8.6 versus 3.8 d; $p < 0.001$) and hospital (median, 19.9 versus 13.2 d; $p = 0.009$) stays. Practice variation existed after stratifying for differences in Glasgow Coma Scale scores (10 versus 7, $p < 0.001$) at time of meeting readiness criteria, particularly for comatose patients. There was a similar reintubation rate. Median hospital charges were $29,057.00 higher for extubation delay patients ($p < 0.001$). This study does not support delaying extubating patients when impaired neurologic status is the only concern prolonging intubation. A randomized trial of extubation at the time brain-injured patients fulfill standard weaning criteria is justifiable.

Measuring Intra-Esophageal Pressure to Assess Transmural Pulmonary Arterial Occlusion Pressure in Patients with Acute Lung Injury: A Case Series and Review

Richard H Kallet MS RRT, Jeffrey A Katz MD, Jean-François Pittet MD, Joy Ghermey MD, Mark Siobal RRT, James A Alonso RRT, and James D Marks MD PhD

BACKGROUND: Positive end-expiratory pressure (PEEP) may interfere with accurate assessment of cardiac function. PEEP may decrease left ventricular volume by lowering the transmural gradient between ventricular and pleural surface pressure (P_{PL}) around the heart while raising the absolute pulmonary arterial occlusion pressure (PAOP). Clinical formulas used to predict the transmural PAOP ($PAOP_{TM}$) require subtracting 25–50% of the PEEP level from the PAOP. However, both PAOP and P_{PL} are influenced by transmitted PEEP and transmitted intra-abdominal pressure (IAP). We compared $PAOP_{TM}$ calculated by measuring intra-esophageal pressure (P_{ES}) with $PAOP_{TM}$ estimated by clinical formulas. METHODS: Twenty-two P_{ES} measurements were made with a bedside pulmonary mechanics monitor (BICORE CP-100) on 11 patients with acute lung injury who had an elevated PAOP (mean ± standard deviation) of 21.1 ± 6.2 mm Hg and PEEP of 13.0 ± 3.8 mm Hg. Paired comparisons were made with the Wilcoxon signed-rank test and multiple comparisons were made using one-way analysis of variance (ANOVA) and the Student-Newman-Keuls test. Pearson product-moment correlation coefficients were calculated. A MEDLINE literature search was done to survey the reported range of PEEP transmitted to P_{PL}. RESULTS: P_{ES} (14.6 ± 5.0 mm Hg) exceeded PEEP; 9 of 11 patients had clinical evidence of increased IAP. $PAOP_{TM}$ predicted by clinical formulas were 13.5–17.7 mm Hg, whereas $PAOP_{TM}$ calculated by P_{ES} was 6.2 ± 3.6 mm Hg ($p < 0.05$). Linear regression revealed a moderate correlation between PAOP and PEEP ($r = 0.49$, $p = 0.02$). In contrast, there was a strong correlation between PAOP and P_{ES} ($r = 0.83$, $p < 0.0001$). A review of data derived from the literature did not show a consistent pattern of PEEP transmission. CONCLUSION: $PAOP_{TM}$ calculated by P_{ES} may reflect transmitted IAP to the pleural surface. Using P_{ES} to calculate $PAOP_{TM}$ may provide a more accurate assessment of hemodynamic status than predicting $PAOP_{TM}$ using clinical formulas based solely on estimated PEEP transmission. *Key words: intra-esophageal pressure, transmural, pulmonary arterial occlusion pressure, acute lung injury, acute respiratory distress syndrome, positive end-expiratory pressure, pleural pressure, abdominal compartment syndrome.* [Respir Care 2000;45(9):1072–1084]

Background

Management of hemodynamic instability during acute respiratory distress syndrome (ARDS) is often complicated by the need for high levels of positive end-expiratory pressure (PEEP) and by the underlying disease process. PEEP may decrease cardiac output by reducing venous return, thereby decreasing left ventricular end-diastolic volume (LVEDV) while raising left ventricular end-diastolic pressure (LVEDP).[1] Decreased cardiac output coupled with

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increased LVEDP is usually suggestive of left heart failure.[2] Yet volume infusion during mechanical ventilation with PEEP generally restores cardiac output.[1,3–5] However, high levels of PEEP may also cause myocardial ischemia.[6] ARDS precipitated by septic shock or pancreatitis generally requires aggressive fluid resuscitation to maintain systemic perfusion,[7–10] despite the fact that cardiac function is frequently impaired.[8,11] Therefore, accurate assessment of left ventricular performance in complex cases of ARDS is often difficult.

The major determinant of left ventricular performance is the LVEDV.[12] If left ventricular compliance is normal, then LVEDP can be substituted for LVEDV.[13] LVEDP is reflected by the left atrial pressure (LAP),[12] which in turn is estimated by measuring the pulmonary arterial occlusion pressure (PAOP) with a pulmonary arterial catheter (PAC). Close agreement between PAOP and LAP requires a patent blood-filled segment of the pulmonary vasculature to serve as an extension of the PAC.[14] However, PEEP can cause capillary collapse when alveolar pressure exceeds capillary pressure distal to the PAC tip,[15–19] so that PAOP may reflect PEEP rather than LAP.

More importantly, the heart is situated within a cavity between the lungs (the cardiac fossa),[20] so that lung inflation affects left ventricular compliance[21] and end-diastolic volume. LVEDV is in part determined by the gradient between LVEDP and the pleural surface pressure (P_{PL}) surrounding the heart (transmural filling pressure).[20,22–24] When high levels of PEEP are required, some believe that only transmural filling pressure can accurately reflect a patient's hemodynamic status[1] and that clinical decisions based on PAOP are risky.[25] Calculating transmural PAOP ($PAOP_{TM}$) requires measuring intra-esophageal pressure (P_{ES}), as it is the only semi-invasive, clinically-available surrogate for mean P_{PL}.[26] This is seldom done because P_{ES} may not reflect mean P_{PL},[26–28] particularly in the cardiac fossa.[29] Instead, some have suggested using clinical formulas to predict PEEP transmission to the pleural surface.[30–33] Yet concerns over the accuracy of P_{ES} must be weighed against the uncertainty of clinical formulas.[34]

Therefore, we questioned whether $PAOP_{TM}$ calculated by P_{ES} provided better clinical information than formulized estimates. We report 11 cases of hemodynamic instability in complex cases of ARDS and acute lung injury (ALI) in which $PAOP_{TM}$ was calculated by P_{ES} and compared to $PAOP_{TM}$ predicted by formulized estimates. In addition, a literature review was done to investigate the reported range of PEEP transmission to the pleural surface during mechanical ventilation.

Materials and Methods

Patients

Between October 1997 and August 1999, P_{ES} measurements for estimation of $PAOP_{TM}$ were requested by the critical care service managing 11 patients who met North American-European Consensus Conference criteria for ARDS/ALI.[35] In these patients, the diagnosis of ARDS/

Table 1. Characteristics of Patients with Acute Lung Injury Requiring Calculation of Transmural Pulmonary Arterial Occlusion Pressure

Case	Sex	Age	Diagnosis and Management Problems
1	M	45	ALI, peritonitis and severe ascites, septic shock, asthma exacerbation, myocardial infarction.
2	M	24	ARDS; multiple orthopedic fractures; splenic, liver, and pulmonary lacerations; abdominal distension; MOSF.
3	M	34	ALI, acute alcoholic pancreatitis, severe ascites, MOSF, metabolic acidosis, asthma exacerbation, bilateral pleural effusions.
4	M	71	ARDS, necrotizing pancreatitis, septic shock, severe ascites (IAP = 33–40 mm Hg), MOSF, bilateral pleural effusions ≥ 1 L found on autopsy.
5	F	55	ARDS, acute myocardial infarction, MOSF.
6	F	75	ARDS, pneumonia and sepsis, with a left-sided pleural effusion.
7	M	52	ARDS, AIDS, pancreatitis, severe bronchospasm, mildly enlarged heart, and abdominal distension.
8	M	73	ARDS, multiple orthopedic fractures, persistent retroperitoneal bleeding, left leg necrosis, pneumonia, septic shock, and MOSF.
9	F	33	ALI, fetal distress (31 weeks gestation), pancreatitis, hypotension, and abdominal compartment syndrome with IAP = 26 mm Hg.
10	M	64	ARDS, sepsis, history of CHF (initial PAOP = 10 mm Hg), ischemic bowel, severe ascites, MOSF.
11	F	47	ARDS, neurogenic pulmonary edema, septic shock, and abdominal distension.

ALI = acute lung injury. ARDS = acute respiratory distress syndrome. MOSF = multiple-organ system failure. IAP = intraabdominal pressure. AIDS = acquired immune deficiency syndrome. CHF = congestive heart failure. PAOP = pulmonary arterial occlusion pressure.

ALI was made either without clinical evidence of increased LAP prior to the placement of the PAC or in conjunction with an initial PAOP measurement of ≤ 18 mm Hg.[35] Medical records were reviewed to abstract pertinent data. Both diagnoses and management problems were summarized (Table 1).

Measurements

To assess $PAOP_{TM}$, a nasogastric tube equipped with an esophageal balloon (Smart Cath, Thermo Resp Group, Palm Springs, California) was passed into the stomach and then withdrawn into the esophagus while inspecting the P_{ES} tracing for the appearance and then minimization of cardiac artifact.[36] The final position of the esophageal balloon was approximately 35–45 cm from the nares, where P_{ES} is approximately 2 mm Hg greater than regional P_{PL} in the supine position.[37] The esophageal balloon was attached to a bedside pulmonary mechanics monitor (BICORE CP-100, Thermo Respiratory Group, Palm Springs, California) and inflated to a volume of 0.8 mL. Because the esophageal balloon is long (10 cm) and the pressure surrounding it is not uniform, the air inside the balloon is displaced to the point of lowest regional pressure.[38] To further minimize mediastinal compression artifact, P_{ES} was measured in the semi-Fowlers (20–30°) position.[39,40] In 6 cases, (1,2,6,9–11), 13 measurements were made comparing P_{ES} between the semi-Fowlers and supine positions. Because some[41] believe that P_{ES} can be accurately measured only in the lateral decubitus position, we compared P_{ES} between the semi-Fowlers and lateral decubitus positions in 4 cases (2, 8, 9, and 11). Because our patients required neuromuscular blockade and/or high levels of sedation, the airway occlusion test[42] for balloon position confirmation could not be done.

Pulmonary mechanics were assessed by measuring the airway plateau pressure (P_{PLAT}) at end-inspiration during a 0.5–1.0 second zero-flow pause. P_{ES} was measured at end-expiration marked from the expiratory flow tracing on the BICORE monitor. In 9 patients, the change in esophageal pressure from end-expiration to end-inspiration (ΔP_{ES}) was also measured from scalar pressure waveforms. Airway pressures (P_{AW}) and tidal volumes (V_T) were measured from the monitoring system of each patient's ventilator (Dräger E-1 and E-2, Telford, Pennsylvania). Intrinsic PEEP ($PEEP_I$) was measured during a 1–2 second end-expiratory pause.[43] Total PEEP ($PEEP_T$) was recorded and represented the sum of set PEEP and $PEEP_I$.[43]

Mean arterial blood pressure (MAP), right atrial pressure (RAP), and PAOP were measured with standard pressure transducers, zeroed to atmospheric pressure, referenced to mid-axillary position, and electronically calibrated. PAOP was measured with a slow injection of 0.1–0.2 mL of air up to 1.5 mL, until both a change in the waveform morphology and a drop in mean pulmonary arterial pressure occurred.[2] Measurements were made at end-expiration. Chest radiographs revealed that PAC tips were properly located in a main branch of the pulmonary artery. Intra-abdominal pressure (IAP) was measured in two patients by the bladder pressure method.[44] All patients were sedated and 9 of 11 patients were receiving neuromuscular blockade when P_{ES} measurements were made. In the other two cases (9 and 10) spontaneous breathing efforts were absent.

Calculations

Delivered V_T from the ventilator was corrected by subtracting the estimated volume loss from circuit compression. Ventilator circuit compliance was 2.5–2.7 mL/cm H_2O. Compression volume loss was calculated as: (peak P_{AW} – set PEEP) × circuit compliance. Quasi-static respiratory system compliance (C_{RS}) and its subcomponents, lung compliance (C_L) and chest wall compliance (C_{CW}), were determined using the following equations adjusted for $PEEP_I$.[45]

$$C_{RS} = V_T \div (P_{PLAT} - PEEP_T) \quad (1)$$

$$C_L = V_T \div [(P_{PLAT} - PEEP_T) - \Delta P_{ES}] \quad (2)$$

$$C_{CW} = V_T \div \Delta P_{ES} \quad (3)$$

Both C_L and C_{CW} determine the amount of PEEP transmitted to the pleural surface,[22,46] and may be predicted from the equation: $C_L \div (C_L + C_{CW})$.[22,46] Jardin et al[47] described how derangements in C_{RS} affected PEEP transmission to the pleural surface. We compared our results of $PAOP_{TM}$ calculated from measuring P_{ES} to predicted $PAOP_{TM}$ using (1) clinical formulas,[31,33] (2) measured values of C_L and C_{CW},[41,46] and (3) the observations of Jardin et al.[47]

$$PAOP_{TM}{}^{31} = PAOP - 50\% \text{ of PEEP} \quad (4)$$

$$PAOP_{TM}{}^{33} = PAOP - 2 \text{ mm Hg per 5 cm } H_2O \text{ PEEP above 10 cm } H_2O \text{ PEEP} \quad (5)$$

$$PAOP_{TM}{}^{46} = PAOP - PEEP_T \times [C_L \div (C_L + C_{CW})] \quad (6)$$

$$PAOP_{TM}{}^{47} = PAOP - 24\text{–}32\% \text{ of PEEP (based on } C_{RS}) \quad (7)$$

Statistical Analysis

Data are reported as means ± standard deviations. Multiple comparisons between the calculated $PAOP_{TM}$ and the various clinical formulas used to predict $PAOP_{TM}$ were made using one-way analysis of variance (ANOVA) and the Student-Newman-Keuls test.[48] Paired comparisons were made using the Wilcoxon signed-rank test.[48] Pearson product-moment correlation coefficients were calculated to determine the strength of association between measurements of PAOP, P_{ES}, and $PEEP_T$.[48] Differences were considered significant if $p < 0.05$.

Literature Review

A MEDLINE search was done for reports published between 1970 and 1999 that investigated cardiovascular response to mechanical ventilation with PEEP. The literature review included both animal and human studies, with both normal lungs and ALI. In order to survey the range of PEEP transmission to the pleural surface, data were abstracted from studies that reported P_{ES}, P_{PL}, epipericardial pressure (P_{EP}), or pericardial pressure (P_{PC}). These data were used in an attempt to clarify 3 questions. First, do changes in P_{ES} reasonably reflect changes in P_{PL}, P_{EP}, and P_{PC} as PEEP is varied? Second, do lung mechanics affect PEEP transmission in a manner predicted by the relationship between C_L and C_{CW} [Equation 6]? Third, because C_L partially determines PEEP transmission, did increasing levels of PEEP affect alveolar pressure transmission to the pleural surface because of improving C_L? PEEP transmission was calculated as the change in either P_{ES}, P_{PL}, P_{EP}, or P_{PC} from ambient pressure to each PEEP level, relative to each PEEP level.

Results

Case Series

The patients had complicated cases of ARDS/ALI, and aggressive fluid resuscitation and/or vasopressor therapy was required to maintain arterial blood pressure and pH in the presence of septic shock and/or pancreatitis. ARDS/ALI originated from extrapulmonary disease in 10 subjects and from pulmonary disease in the other case.[49] Ten of the 11 patients had clinical evidence of chest wall restriction (abdominal distention, severe ascites, or pleural effusions) and 3 patients also had severe bronchospasm (see Table 1). Mean (± standard deviation) C_{RS} was 26 ± 8 mL/cm H_2O, and there was no significant difference between C_L (57 ± 25 mL/cm H_2O) and C_{CW} (59 ± 22 mL/cm H_2O) ($p = 0.74$). However, repeated measurements in 4 patients (1, 5, 10, and 11) revealed pronounced variability in C_L and C_{CW} (Fig. 1). C_L and C_{CW} sometimes changed both in direction and magnitude without an appreciable change in the corresponding C_{RS} (Fig. 2). With the exception of 4 discreet measurements in cases 5, 9, and 10, all patients had measurable $PEEP_I$.

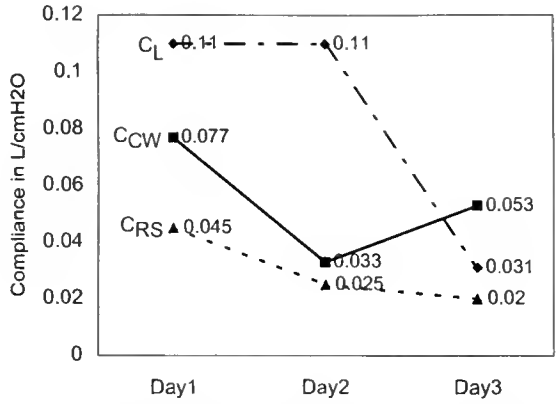

Fig. 1. Variability in the compliance of the lung (C_L), chest wall (C_{CW}), and respiratory system (C_{RS}) over time in a patient with an extrapulmonary source of ARDS (case 5).

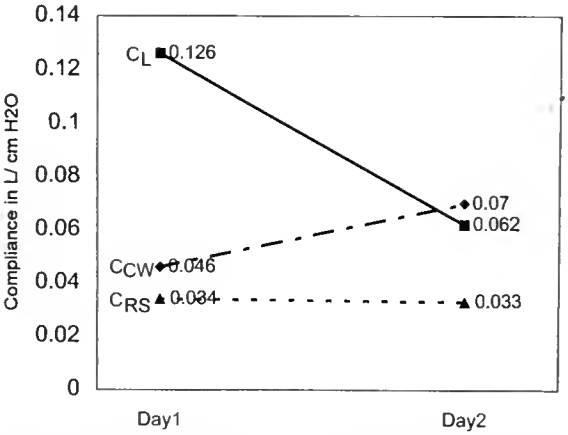

Fig. 2. Stability in respiratory system compliance (C_{RS}) with pronounced variability in its subcomponents, lung compliance (C_L) and chest wall compliance (C_{CW}), over time in a patient with an extrapulmonary source of acute respiratory distress syndrome (case 11).

PAOP was greater than the reported range for ARDS (9–18 mm Hg).[32] Yet, the calculated $PAOP_{TM}$ was significantly lower ($p < 0.0001$) and generally was within the normal limits of PAOP during atmospheric breathing (4–12 mm Hg)[50,51] (Table 2). Mean P_{ES} was greater than $PEEP_T$. Therefore, all predicted[31,33,46,47] values of $PAOP_{TM}$ were significantly higher than calculated $PAOP_{TM}$ ($p < 0.05$) (Table 3). Linear regression revealed a moderate, positive correlation between PAOP and $PEEP_T$ ($r = 0.49$, $p = 0.02$) and between P_{ES} and $PEEP_T$ ($r = 0.61$, $p = 0.003$). In contrast, there was a strong positive correlation between PAOP and P_{ES} ($r = 0.83$, $p < 0.0001$) (Fig. 3).

Table 2. Comparison of Cardiac Filling Pressures Referenced to Atmospheric Pressure Versus Transmural Filling Pressures

	RAP (mm Hg)	PAOP (mm Hg)	$PEEP_T$ (mm Hg)	P_{ES} (mm Hg)	RAP_{TM} (mm Hg)	$PAOP_{TM}$ (mm Hg)
Mean ± SD	21.3 ± 8.5	21.1 ± 6.2	13.0 ± 3.8	14.6 ± 5.0	6.7 ± 5.2	6.5 ± 3.5

RAP = right atrial pressure. PAOP = pulmonary arterial occlusion pressure. $PEEP_T$ = total positive end-expiratory pressure (set + intrinsic). P_{ES} = esophageal pressure. TM = transmural. RAP_{TM} = RAP − PES. $PAOP_{TM}$ = PAOP − PES.

Table 3. Comparisons Between Transmural Pulmonary Arterial Occlusion Pressures Calculated by Esophageal Pressure and Transmural Pulmonary Arterial Occlusion Pressures Predicted by Clinical Formulas

	$PEEP_T$ (mm Hg)	P_{ES} (mm Hg)	PAOP (mm Hg)	$PAOP_{TM}$ (mm Hg)	$PAOP_{TM}$[33]* (mm Hg)	$PAOP_{TM}$[47]† (mm Hg)	$PAOP_{TM}$[31]‡ (mm Hg)	$PAOP_{TM}$[22]§ (mm Hg)
Mean ± SD	13.0 ± 3.8	14.6 ± 5.0	21.1 ± 6.2	6.5 ± 3.5	17.7 ± 5.8	17.6 ± 6.0	13.9 ± 5.7	13.6 ± 5.3

$PEEP_T$ = total positive end-expiratory pressure (applied + intrinsic). P_{ES} = esophageal pressure. PAOP = pulmonary arterial occlusion pressure. TM = transmural.
*$PAOP_{TM}$ = −2 mm Hg/5 cm H_2O PEEP above 10 cm H_2O.
†$PAOP_{TM}$ = 24–32% PEEP transmitted based on respiratory system compliance.
‡$PAOP_{TM}$ = 50% PEEP transmission.
§$PAOP_{TM} = PAOP - PEEP_T \times [C_L \div (C_L + C_{CW})]$.

The semi-Fowler's position appeared to lessen mediastinal compression on the esophageal balloon, as the mean P_{ES} was 2.1 ± 2.2 mm Hg lower in the semi-Fowler's than in the supine position ($p = 0.01$). Comparisons between P_{ES} measured in the semi-Fowler's and lateral decubitus positions did not show P_{ES} in the lateral decubitus position to be consistently lower. In cases 2 and 9, the P_{ES} was 1–2 mm Hg lower in the lateral decubitus position. In case 8, P_{ES} was not different, while in case 11, P_{ES} was 3 mm Hg higher in the lateral decubitus position. The mean difference in P_{ES} between positions was zero.

In cases 1 and 4, PAOP was markedly elevated (30 and 34 mm Hg, respectively), and two-dimensional transesophageal echocardiography was performed shortly after P_{ES} measurements. Both patients had severe ascites with abdominal compartment syndrome (ACS).[52] In case 1, the calculated $PAOP_{TM}$ was 13 mm Hg and echocardiography revealed hyperdynamic ventricles with slightly enlarged chamber dimensions. Over several hours, 3.5 L of fluid were removed with continuous venovenous hemofiltration. Repeated measurements revealed that PAOP had decreased from 30 to 24 mm Hg at the same P_{ES} (17 mm Hg), resulting in a calculated $PAOP_{TM}$ of 7 mm Hg. In case 4, the calculated $PAOP_{TM}$ was 12 mm Hg. However, the MAP persistently fell below 80 mm Hg despite intravenous fluid administration of 250 mL/h and frequent large boluses of plasmalyte and albumin. The patient's IAP was 33–40 mm Hg, which caused large amounts of ascitic fluid transudation from the abdominal wound. By the next day, the PAOP had decreased to 22 mm Hg and the calculated $PAOP_{TM}$ fell to 1 mm Hg. This coincided with a decreased urine output, from 86 to 16 mL/h, and a drop in mixed venous oxygen saturation, from 67% to 58%. Echocardiography revealed near-collapse of the inferior vena cava during inspiration, and almost complete emptying of the left ventricle at end-systole. In case 11, transthoracic echocardiography was performed after the P_{ES} measurement. The PAOP was 22 mm Hg, whereas the calculated $PAOP_{TM}$ was 10 mm Hg. Echocardiography revealed normal left ventricular function and volume status.

Literature Review

Forty-five papers were reviewed that either directly studied the cardiovascular effects of PEEP[1,3–5,25,29,40,53–87] or studied PEEP transmission under various conditions of lung mechanics.[22,46,47] Data could be abstracted from 41 studies. Ten studies compared P_{ES} with either P_{EP} or P_{PC} as PEEP was varied.[22,53,56,59,61–63,67,69,82] P_{ES} tended to underestimate or overestimate PEEP transmission to P_{EP} or P_{PC}, depending on body position, lung mechanics, and volume status. In normal lungs, both Marini et al[62] and Craven and Wood[61] found that P_{ES} in the lateral decubitus position overestimated P_{EP} by 4–6%, whereas Prewitt and Wood[67] reported that P_{ES} underestimated P_{EP} by 6–15%. Marini et al[62] and O'Quin et al[22] reported that, in normal lungs, P_{ES} in the supine position underestimated PEEP transmission to P_{EP} by 5–9%, whereas Cassidy et al[63] found < 1 cm H_2O difference between mean P_{ES} and P_{PC}. In ARDS, Dhainaut et al[56] reported that P_{PC} exceeded P_{ES} in the supine position by 4 mm Hg at 20 cm H_2O PEEP. Data from Bonnet et al[59] showed that P_{ES} underestimated P_{PC} by 2 mm Hg in cardiac surgery patients with near-normal lung mechanics, but P_{ES} overestimated P_{PC} by 2 mm Hg in patients with pulmonary edema. In normal lungs, Kingma et al[82] found that P_{ES} in the supine position con-

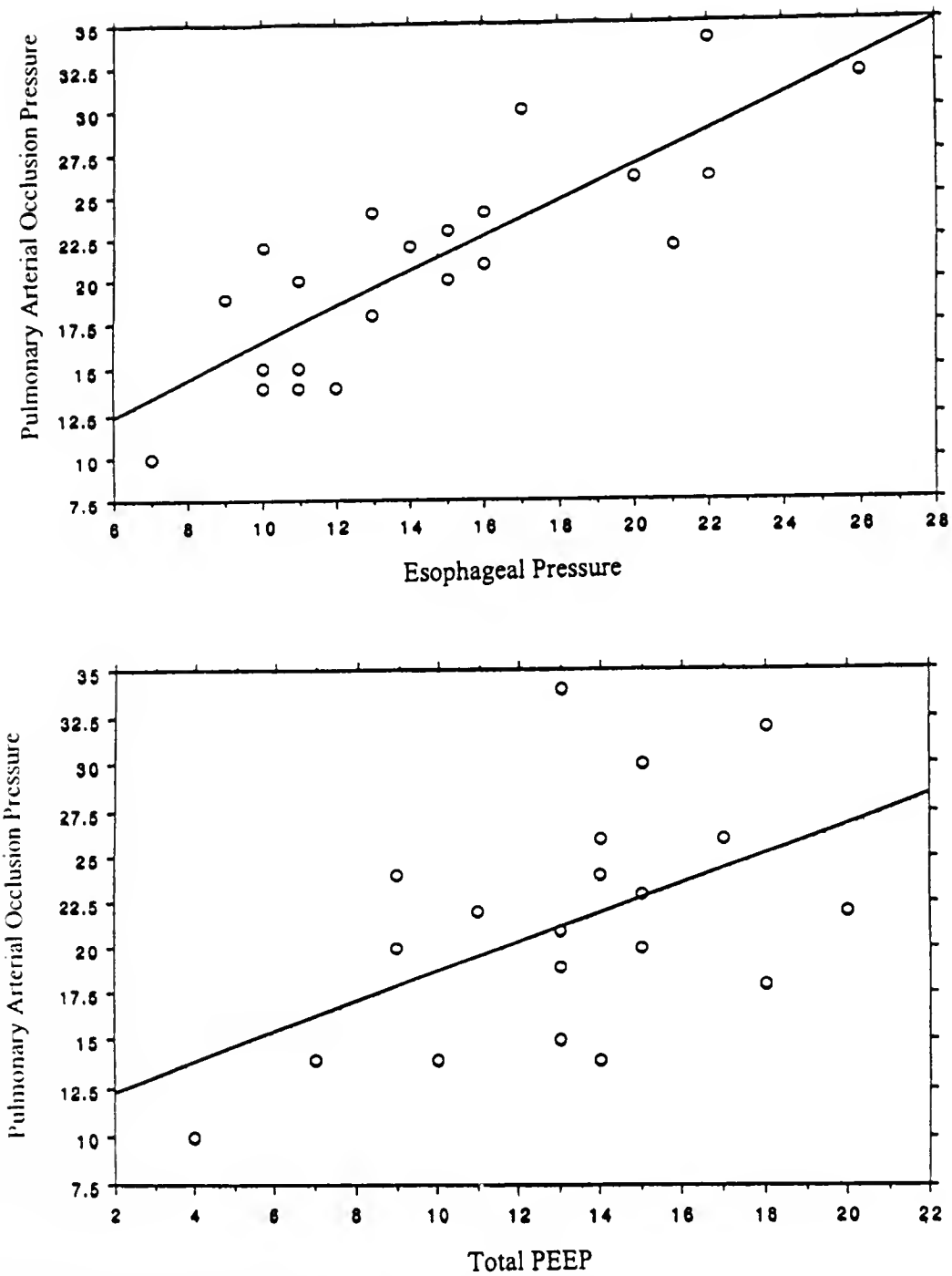

Fig. 3. Linear regression plots of (top) pulmonary arterial occlusion pressure versus esophageal pressure ($r = 0.83$, $p < 0.0001$) and (bottom) pulmonary arterial occlusion pressure versus total positive end-expiratory pressure (PEEP) ($r = 0.49$, $p = 0.002$). All variables are expressed in mm Hg.

sistently underestimated P_{EP} by 3–12 mm Hg, with the discrepancies widening as both PEEP and intravascular volume increased.

Both C_L and C_{CW} determine PEEP transmission to the pleural surface.[22,46] Data on PEEP transmission with normal lungs was reported in 27 studies. In 16 of those studies,[3,22,29,46,53,54,58,64–68,78,80,82,85] between 42% and 59% of the PEEP was reflected in either the P_{ES}, P_{PL}, P_{EP}, or P_{PC}. Yet, under some study conditions, PEEP transmission deviated markedly from expected: 61–75%,[1,22,25,58,61,67,82,84]

and 20–40%.[4,5,29,54,63,72,73,75,76,78,79,87] More PEEP should be transmitted to the pleural surface when C_L is higher than C_{CW}, and less when C_L is lower than C_{CW}. Chapin et al[46] and O'Quin et al[22] conducted the only rigorous studies on lung mechanics and PEEP transmission, but their results indicated widely variable PEEP transmission, depending on measurement technique. When C_L (or C_L and C_{CW}) was reduced, lateral P_{PL}[46] underestimated PEEP transmission relative to P_{EP}[22] (30% and 55%, respectively). Decreased C_{CW} alone consistently caused increased PEEP transmission (64–80%).[22,46,66] In other ALI animal studies, PEEP transmission was either $< 40\%$[29,58,61,63,72] or $> 40\%$.[65,67] Data from human studies of ALI/pulmonary edema also showed inconsistent PEEP transmission that was either $< 40\%$[47,56,70,74] or approximately 50%.[40,59] In one study where C_L was low and PEEP transmission was reduced, C_{CW} was usually above 100 mL/cm H_2O.[47]

PEEP transmission during ARDS/ALI may change as PEEP is altered. If increasing PEEP improves C_L, it may also increase airway pressure transmission to the pleural surface.[77] Twenty-four studies reported data when PEEP was varied by 10–15 cm H_2O under conditions of both normal lungs and ALI/pulmonary edema. In 16 studies, PEEP transmission increased 6–48%.[3,4,18,29,56,57,59,60,61,63,70,74,76,79,82,87] In summary, data derived from the literature suggest that:

1. P_{ES} tends to underestimate P_{EP}/P_{PC}, with the differences generally within 10% or 1–3 mm Hg;
2. PEEP transmission during normal and ALI conditions is generally between 40% and 60% (although transmission often exceeded this range); and
3. PEEP transmission tends to increase with increasing amounts of PEEP.

The variety of study subjects/models, measurement techniques, and pressure-sensing devices used among the studies limits the ability to generate strong conclusions.

Discussion

There are 5 findings from the case series and the literature review. First, echocardiography studies in three of our patients with ARDS/ALI suggested that elevated PAOP was not necessarily indicative of either intravascular volume overload or cardiac failure. In cases 4 and 11, the PAOP was 22 mm Hg and echocardiography revealed either intravascular volume depletion (case 4) or normal left ventricular function and volume status (case 11). In case 1, where the PAOP was 30 mm Hg, echocardiography demonstrated intact cardiac function with only modest intravascular volume overload. Our results support Downs's[88] observation that a PAOP > 20 mm Hg is often necessary to maintain cardiac output in severe ARDS.

Second, $PAOP_{TM}$ predicted by clinical formulas greatly exceeded $PAOP_{TM}$ calculated by P_{ES}. Normally, C_L and C_{CW} are approximately equal in the V_T range,[89] so that 50% of the PEEP level should be reflected in measurements of P_{PL}. Boysen[31] stated that 30–70% of PEEP is transmitted to the pleural surface, and subtracting 50% of PEEP from PAOP was used for clinical convenience. Goldenheim and Kazemi's[33] clinical formula is based on evidence that PEEP transmission is clinically negligible at $\leq$ 10 cm H_2O.[18,90] Subtracting 2 mm Hg/5 cm H_2O of PEEP above 10 also implies 50% transmission (eg, 2/3.67 mm Hg = 0.54). Predicted $PAOP_{TM}$ based on C_L and C_{CW} also overestimated $PAOP_{TM}$ calculated by P_{ES}. In the literature review, PEEP transmission did not consistently conform to theoretical predictions based on C_L and C_{CW}, and large variations in transmission occurred even when normal lungs were studied. Predicting PEEP transmission based on C_{RS} may be more uncertain. As shown in some of our patients with extrapulmonary etiologies of ARDS, C_L and C_{CW} were not stable, but changed both in magnitude and direction, even when C_{RS} was unaltered.

Third, data derived from 67% of the relevant studies indicated that PEEP transmission rose as PEEP increased, suggesting the possibility that lung recruitment may alter PEEP transmission to the pleural surface. Therefore, predicting $PAOP_{TM}$ according to fixed levels of PEEP transmission may cause overestimation of actual $PAOP_{TM}$. Extrapulmonary etiologies of ARDS are more responsive to lung recruitment (because of decreased C_{CW} and atelectasis),[49,91] so that these patients are more prone to hemodynamic deterioration with increasing PEEP.[49] This is supported by Kotanidou et al,[92] who found that increasing PEEP decreased cardiac index only when C_{RS} increased.

Fourth, P_{ES} exceeded $PEEP_T$ in our subjects, most of whom had evidence of increased IAP. Therefore, factors other than PEEP that contribute to P_{PL} can only be accounted for by measuring either P_{ES} or P_{PL}.

Fifth, some of the reviewed studies indicated that P_{ES} may reflect changes in P_{EP}/P_{PC} reasonably well for clinical purposes.

Calculating $PAOP_{TM}$ with P_{ES} is limited by balloon stress artifact that causes P_{ES} to overestimate mean P_{PL}.[93] The most important causes of balloon stress are mediastinal compression[94] and esophageal muscle tension.[95,96] In the supine position, the heart impinges directly on the esophageal balloon, causing P_{ES} to exceed local P_{PL}, especially at low lung volumes.[27,97] Our balloons were positioned 35–45 cm from the nares, which should have placed them in the more compliant[96] mid-to-lower third of the esophagus (Fig. 4).[98,99] At this position, P_{ES} is relatively uniform[98] because the air in long balloons (10 cm) is displaced to the point of lowest regional pressure.[99] Mediastinal compression increases P_{ES} by approximately 4 mm Hg, compared to both the sitting position[27,28,97] and to adjacent P_{PL}.[95] The lateral decubitus position improves P_{ES} accuracy,[41,98] as values of –2 mm Hg are reported at

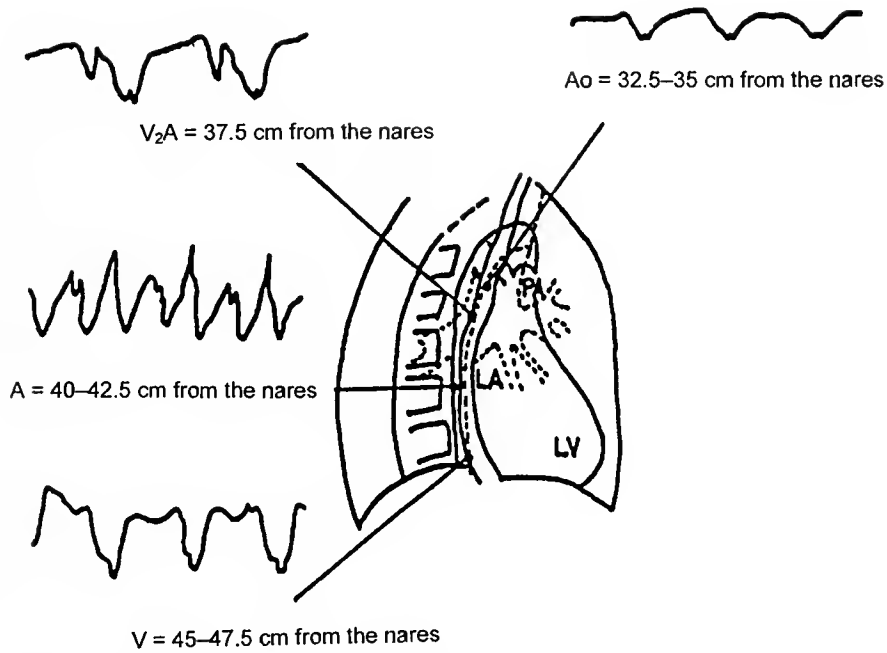

Fig. 4. Schematic drawing of the thorax, illustrating the anatomic relationship between the depth (from the nares) of the esophageal balloon and its position next to the heart. The pressure waveforms represent the typical cardiac artifacts encountered at different levels of the heart during spontaneous breathing. The terms V2A, Ao, A, and V refer to the pressure waveforms seen at different depths in the esophagus. (Modified from Reference 99, with permission.)

ambient pressure.[61] Yet, P_{ES} may underestimate P_{PL} in the cardiac fossa, because lung inflation with PEEP tends to both compress the heart[20] and lift the heart off of the esophagus.[62] In some studies, P_{ES} underestimated P_{EP} by approximately 2 mm Hg[22,62] and P_{PC} by 4 mm Hg.[56] Therefore, the error caused by mediastinal compression artifact may be partially offset by an opposing error in which P_{ES} underestimates P_{PL} in the cardiac fossa.

P_{ES} equaled or exceeded $PEEP_T$ in 68% of our measurements. Some of this aberrancy was probably due to P_{ES} artifact and underestimation of $PEEP_I$ when measured by the circuit occlusion method.[100] However, 9 of 11 patients had abdominal distention or ascites. Clinicians are accustomed to thinking exclusively in terms of how PEEP affects P_{PL}. But increased IAP occurs in approximately 20–30% of the medical/surgical intensive care population,[101] as a consequence of intra-abdominal/retroperitoneal hemorrhage, ascites, bowel distention, and placement of intra-abdominal packs.[102] Increased IAP may cause the diaphragm to protrude into the thoracic cavity, thus raising intrathoracic pressure[103] and decreasing myocardial compliance.[104] Sustained high IAP decreases venous return and cardiac output while raising RAP, LAP, and P_{PL}.[52,101–107] Consequently, transmural filling pressures decrease.[102,103] In our patients, mean RAP and PAOP were not different ($p = 0.92$), suggesting that the combination of high PEEP and IAP may create an intrathoracic tamponade.

PAOP correlated more highly with P_{ES} ($r = 0.83$) than it did with $PEEP_T$ ($r = 0.49$). This suggests that P_{ES} and PAOP were reflecting forces other than PEEP that contributed to P_{PL}. In our case series, ARDS/ALI occurred with sepsis, pancreatitis, pleural effusions, and ACS, which can increase P_{PL} as well as enhance P_{ES} artifact. Sepsis produces generalized microvascular injury,[108] resulting in "whole body inflammation."[109] Pulmonary edema increases P_{ES} because of increased lung weight.[63] Therefore, lung, chest wall, and myocardial edema may raise intrathoracic hydrostatic pressure and increase both P_{ES} and P_{PL}. ACS causes the diaphragm to shift cephalad,[101] resulting in a posteriolateral displacement of the heart and esophageal compression.[27] Therefore, an otherwise properly positioned esophageal balloon may be placed within the supradiaphragmatic but functional intra-abdominal compartment [JJ Marini, University of Minnesota, personal communication, 2000]. However, because ACS compresses the dependent lung, cardiac fossa compliance is also decreased[104] and transmural filling pressures are reduced.[101] In two of our ACS cases, transesophageal echocardiography verified the intravascular volume status suggested by the calculated $PAOP_{TM}$.

The alternative method for assessing cardiac filling pressures has been to measure PAOP after disconnecting the patient from the ventilator for 5–15 seconds.[110,111] In severe ARDS, abruptly removing high levels of PEEP car-

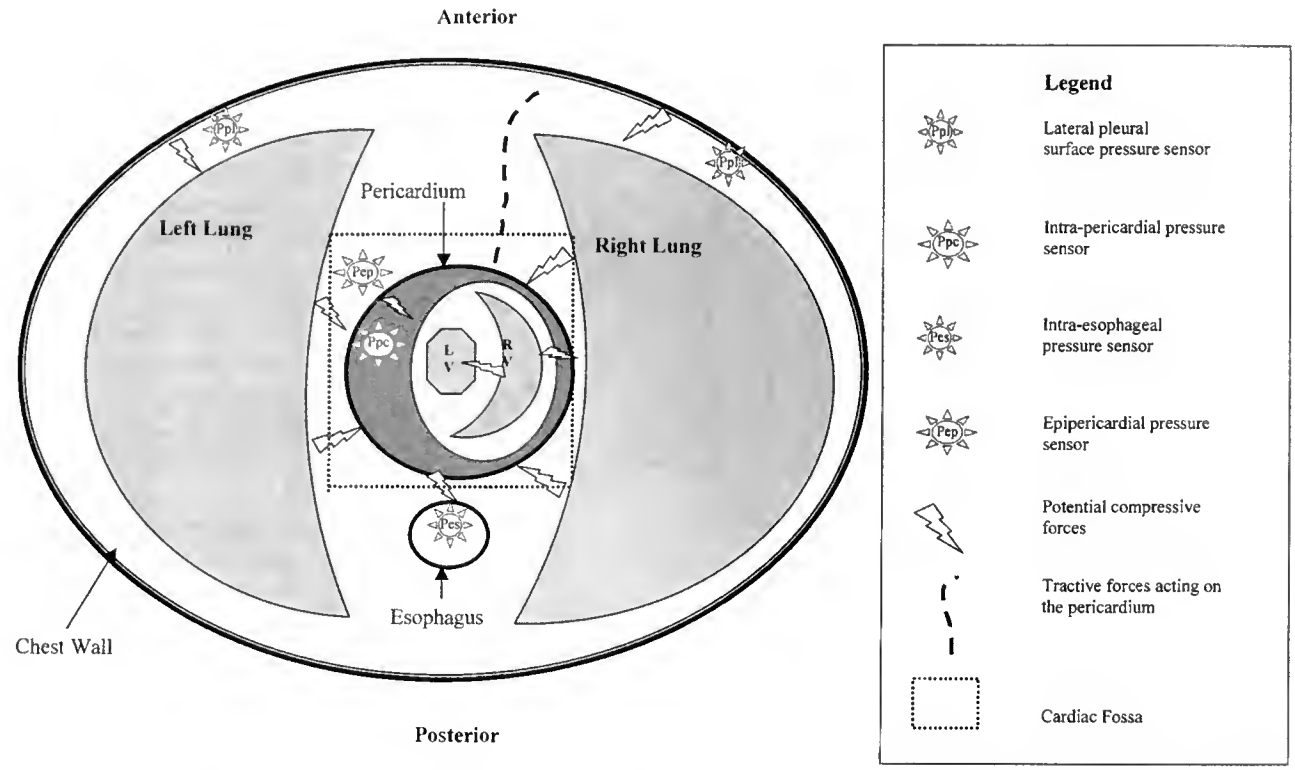

Fig. 5. Schematic drawing of the thorax, illustrating the cardiac fossa, the orientation of various intrathoracic pressure transducers, and potential compressive forces acting on intrathoracic structures. LV = left ventricle. RV = right ventricle.

ries the risk of acute alveolar edema[111] and hypoxemia.[112] Interpreting the PAOP measured off-PEEP is difficult because the accompanying changes in blood volume distribution and pressure may not correspond to PEEP conditions,[112] and the PAOP and P_{PL} may not decrease uniformly.[111] Carter et al[25] reported that the immediate drop in PAOP after abrupt ventilator disconnection ("nadir wedge") represents the change in P_{PL} with PEEP and may be rapid enough (2–3 s)[57] to precede significant changes in venous return. In an animal model of ALI, the nadir wedge accurately reflected transmural LAP only when the latter was < 10 mm Hg.[25] In postoperative cardiac surgery patients, nadir wedge accurately reflected transmural LAP up to 15 cm H_2O of set PEEP.[57] However, the nadir wedge will not account for the effects of $PEEP_I$[113] and IAP on P_{PL}. An additional uncertainty is that the sudden decrease in lung volume with ventilator disconnection may cause a rapid shift in blood volume from the pulmonary circulation to the left heart.[113] This may result in a falsely elevated transmural LAP.

Assessing the cardiovascular effects of mechanical ventilation with PEEP is further complicated by the following variables: lung volume,[22] regional differences in lung volume,[114] V_T size,[23] and V_T distribution pattern.[55] Lloyd[114] has shown that increased lung volume can restrain pericardial expansion *independent of its effect on regional* P_{PL}, (ie, cardiac fossa compliance is shape dependent). Butler et al[115] observed that in obstructive lung disease PAOP could rise markedly with increasing left lower lobe volume, but without an increase in P_{ES}. Marini et al[55] reported that ventilation patterns causing marked lung distention over the right heart were associated with the largest reductions in stroke volume because of decreased venous return. Wallis et al[23] suggested that the combined compressive forces of high V_T and PEEP during inspiration may have a greater impact on left ventricular function than events during expiration. Therefore, evaluating the cardiovascular effects of mechanical ventilation must take into account V_T size and regional lung inflation as well as PEEP.

PAOP is an imprecise approximation of LVEDV in ARDS/ALI. PAOP reflects LVEDV only when left ventricular compliance is normal[116] and in the absence of mitral stenosis[116] and pulmonary venous obstruction.[41] Decreased left ventricular compliance may occur in ARDS[117] and septic shock,[118] for several reasons, including myocardial ischemia and edema,[104] increased myocardial elasticity resulting from shock,[119] increased right ventricular end-diastolic volume,[120] decreased pericardial elasticity,[121] pulmonary compressive forces,[114] and nonpulmonary tractive forces[122] within the cardiac fossa (Fig. 5). Both PEEP[1] and ARDS[123] increase pulmonary vascular resistance, which may cause right ventricular dilation,[120] particularly

under ischemic conditions.[80] This can decrease left ventricular compliance by increasing global pericardial stress[121] and by shifting the interventricular septum toward the left ventricle.[54,124] Furthermore, cardiac fossa compliance declines when either pericardial or thoracic volume rises, because the lungs and chest wall resist deformation more than is assumed by traditional measures of pulmonary mechanics.[114] Therefore, increased heart and lung volume may decrease cardiac fossa compliance, causing P_{PC} to exceed P_{ES}, P_{PL}, and P_{EP}.[82] As a result, $PAOP_{TM}$ calculated by P_{ES} may overestimate the "true" transmural pressure at the ventricular surface.[79,82,122]

In addition to the problem of determining the effect of PEEP on cardiac fossa compliance, the presence of PEEP-artifact due to PAC tip location also must be considered. PAOP may not reflect LAP and LVEDV when the PAC tip is not located in a West Zone III (below the horizontal plane of the left atrium), where pulmonary venous pressure exceeds alveolar pressure.[125] In this situation, the PAOP may reflect PEEP rather than LAP.[15,16,19] Shasby et al[17] found a 43% incidence of PAC tips located in non-West Zone III. Teboul et al[126] reported that West Zone placement of a PAC tip can be determined by comparing the changes in PAOP and pulmonary arterial pressure with a sudden increase in PEEP. Changes in pulmonary arterial pressure should reflect changes in P_{PL}.[126] Therefore, the ratio of change between PAOP and pulmonary arterial pressure with a change in PEEP should be close to 1.0 (if the PAC tip is located in West Zone III). Ratios greater than 1.2 were observed when the PAC tip was placed in upper lung zones.[126] In severe ARDS, 20–30 cm H_2O of PEEP have been applied with little discrepancy between PAOP and LAP when the tip of the PAC is below the level of the left atrium.[16,127] However, PEEP artifact can affect PAOP despite appropriate PAC tip placement during hypovolemia.[19] This may go unrecognized in a patient with continuous fluid extravasation, as progressive intravascular volume depletion may cause the pulmonary vascular segment serving the PAC tip to shift away from a West Zone III.[41] Agreement between PAOP and LAP can be improved by placing the patient in the lateral decubitus position, so that the PAC tip is directed toward the dependent lung, thus creating West Zone III conditions.[128] In our patients, PAOP only correlated moderately with PEEP, which may suggest that most of our PAC tips were located in a West Zone III.

The complexity of cardiothoracic relationships in patients with ARDS/ALI renders clinical decision-making based on hemodynamic data vulnerable to error. Recently, PAC use in critically ill patients has been associated with a higher incidence of mortality.[129–134] Mortality does not appear to be related to either the severity of illness or to complications directly related to PAC placement.[131] Therefore, the higher mortality rates may be partially related to misinterpretation of PAC data that leads to inappropriate therapeutic decision-making.[132–134] Our case series provides some preliminary evidence suggesting that $PAOP_{TM}$ calculated from P_{ES} measurements provides more useful information than formulized estimates of $PAOP_{TM}$, at least in severe extrapulmonary etiologies of ARDS. However, this case series is limited both by the nature of making uncontrolled measurements in clinically unstable patients and by incomplete data. Given these concerns, the efficacy of bedside P_{ES} measurements to accurately assess $PAOP_{TM}$ should be tested prospectively and verified with transesophageal echocardiography.

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The Effects of Pressure Control Versus Volume Control Assisted Ventilation on Patient Work of Breathing in Acute Lung Injury and Acute Respiratory Distress Syndrome

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BACKGROUND: Patient work of breathing (WOB) during assisted ventilation is reduced when inspiratory flow ($\dot{V}_I$) from the ventilator exceeds patient flow demand. Patients in acute respiratory failure often have unstable breathing patterns and their requirements for $\dot{V}_I$ may change from breath to breath. Volume control ventilation (VCV) traditionally incorporates a pre-set ventilator $\dot{V}_I$ that remains constant even under conditions of changing patient flow demand. In contrast, pressure control ventilation (PCV) incorporates a variable decelerating flow wave form with a high ventilator $\dot{V}_I$ as inspiration commences. We compared the effects of flow patterns on assisted WOB during VCV and PCV. METHODS: WOB was measured with a BICORE CP-100 monitor (incorporating a Campbell Diagram) in a prospective, randomized cross-over study of 18 mechanically ventilated adult patients with acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). Tidal volume, inspiratory time, and mean ventilator $\dot{V}_I$ were constant in each mode. RESULTS: At comparable levels of respiratory drive and minute ventilation, patient WOB was significantly lower with PCV than with VCV (0.59 ± 0.42 J/L vs 0.70 ± 0.58 J/L, respectively, $p < 0.05$). Ventilator peak $\dot{V}_I$ was significantly higher with PCV than with VCV (103.2 ± 22.8 L/min vs 43.8 L/min, respectively, $p < 0.01$). CONCLUSIONS: In the setting of ALI and ARDS, PCV significantly reduced patient WOB relative to VCV. The decrease in patient WOB was attributed to the higher ventilator peak $\dot{V}_I$ of PCV. *Key words: acute lung injury, acute respiratory distress syndrome, assisted mechanical ventilation, central respiratory drive, constant flow pattern, decelerating flow pattern, inspiratory flow rate, pressure-time product, pressure control ventilation, volume control ventilation, work of breathing.* [Respir Care 2000;45(9):1085–1096]

Background

Patient work of breathing (WOB) during assisted ventilation is reduced when inspiratory flow ($\dot{V}_I$) from the ventilator exceeds patient flow demand.[1–4] Because patient flow demand reflects inspiratory muscle contractile velocity,[5,6] the ventilator will impose resistive work whenever patient flow demand exceeds ventilator $\dot{V}_I$. Also, during acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), elastic WOB increases because the inspiratory muscles strain against poorly compliant lungs and chest wall.[7,8] Therefore, high ventilator $\dot{V}_I$ delivery also may lessen a patient's elastic WOB by displacing the chest faster than can be achieved by the inspiratory muscles straining under high elastic loads.

Because patients in acute respiratory failure often have unstable breathing patterns, their requirements for tidal volume (V_T) and flow can change from breath to breath.[9]

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Volume control ventilation (VCV) traditionally delivers a breath with a pre-set ventilator $\dot{V}_I$ that remains constant (ie, a rectangular flow waveform),[10] even under conditions of changing patient flow demand. During pressure control ventilation (PCV), peak airway pressure is sustained by a variable, decelerating flow wave form with a high ventilator $\dot{V}_I$ as inspiration commences.[10] Because PCV regulates ventilator $\dot{V}_I$ to maintain a set airway pressure, transmitted negative pressure from patient effort should cause the ventilator $\dot{V}_I$ to increase and to be sustained when patient effort is prolonged. Therefore, the ability of PCV to produce a high, variable, and sustained $\dot{V}_I$ should adapt better to changes in patient flow and V_T requirements. Furthermore, the high $\dot{V}_I$ and decelerating flow pattern of PCV cause a larger portion of the V_T to be delivered earlier during inspiration.[10] This may also reduce the elastic WOB by displacing the chest faster than can be achieved by the inspiratory muscles. Based on those attributes we predicted that PCV would reduce patient WOB during assisted ventilation more than constant flow VCV in patients with ALI and ARDS.

Materials and Methods

Study Subjects

A convenience sample of 18 nonconsecutive ventilator-dependent patients on the medical, general surgery, and trauma surgery services were entered into the study. Signed, informed consent was obtained from either the patient or a relative. The Committee on Human Research at the University of California, San Francisco, approved the study. Patients were either clinically stable, or in the recovery phase of illness when weaning from mechanical ventilation was taking place. Entrance criteria were: minute ventilation ($\dot{V}_E$) $\geq$ 10 L/min, static respiratory system compliance $\leq$ 50 mL/cm H_2O, and meeting the North American-European Consensus Conference definition for either ALI or ARDS.[11] Patients with primary respiratory failure from ALI or ARDS who had an underlying component of chronic obstructive pulmonary disease or asthma also were entered into the study.

Study Protocol

The study incorporated a random presentation of breathing modes in a short, time-series, cross-over design. A blind envelope pull was used to prevent presentation bias. Nine patients were randomized to each presentation order (VCV to PCV and PCV to VCV). Ten minutes were allowed for adaptation and stabilization of breathing pattern between modes. Data collection required approximately 30 minutes in each mode. Afterwards, patients with a positive end-expiratory pressure (PEEP) of $\leq$ 10 cm H_2O were given a short trial of spontaneous breathing on 100% oxygen through a modified Jackson-Reese circuit. Depending on patient tolerance, data were collected once or twice over 1–2 minutes. Tolerance was defined as $<$ 20% change in either heart rate or blood pressure from study conditions and arterial oxygen saturation (as measured via pulse oximetry [S_{pO_2}]) of $\geq$ 95%. This data provided an approximation of patient WOB, $\dot{V}_I$, V_T, and inspiratory time (T_I) in order to compare ventilator settings during assisted ventilation with patients' spontaneous breathing patterns.

Procedures

Upon enrollment into the study, a Smart Cath (Thermo Respiratory Group, Palm Springs, California) nasogastric tube with an esophageal balloon was placed in the lower third of the esophagus. We manipulated the positon of the balloon while inspecting the synchrony of the peak esophageal pressure (ΔP_{ES}) and airway pressure deflections at moderately low trigger sensitivity settings (–5 cm H_2O). When cardiac artifact was minimized, an occlusion test was performed for position confirmation using the method described by Baydur.[12] All patients were studied in the semi-recumbent position. Maximum inspiratory pressure (MIP) was measured using the "Method I" described by Marini et al, in which the airway is occluded at end-expiration, allowing no movement of air in either an inspiratory or expiratory direction.[13] This method was chosen in order to measure muscle strength from approximately functional residual capacity.

Prior to data collection, relaxed chest wall compliance (C_{CW}) curves were constructed using an analysis of 2–5 breaths during a short period of controlled ventilation. This was achieved following additional sedation with a 100–200 μg dose of Fentanyl (in 50 μg doses) to suppress spontaneous respiratory activity. C_{CW} curves were constructed from esophageal pressure-volume tracings with a counterclockwise movement, a narrow loop and a rightward rotation of the axis (Fig. 1).[14] In 3 patients, controlled ventilation could not be achieved and a normal C_{CW} line was used (200 mL/cm H_2O). After patients recovered and began to trigger the ventilator, central respiratory drive and patient effort were monitored until the presedation baseline had been achieved.

Equipment

Two ventilators were used for this study: the Siemens Servo 900C (Siemens Medical Systems Inc, Danvers, Massachusetts) and the Hamilton Veolar (Hamilton Medical Inc, Reno, Nevada). Each ventilator was calibrated prior to the study. The ventilator was set to achieve a T_I of 1.0 second. This was verified by measurement from the scalar $\dot{V}_I$ wave form. The control rate (f) on the ventilator was set between 10 and 12 breaths per minute and was held con-

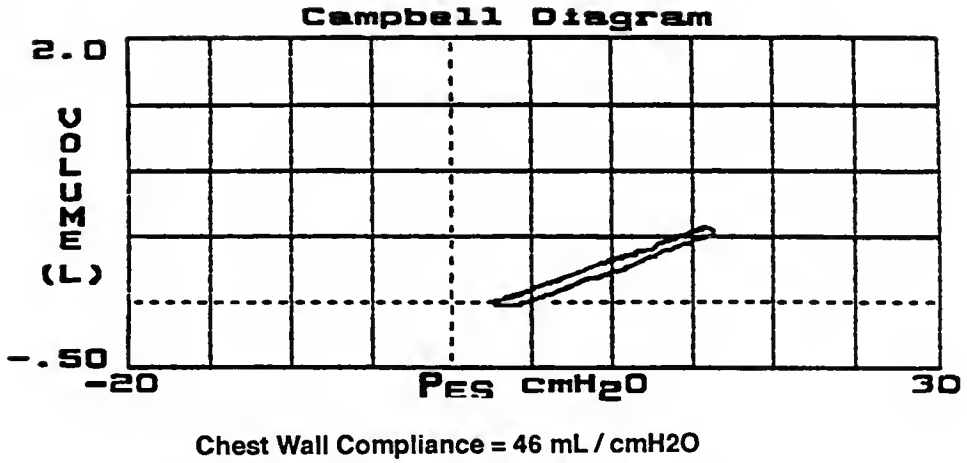


Fig. 1. Campbell diagram for calculation of passive chest wall compliance. Relaxed chest wall compliance is calculated from the esophageal pressure-volume loop. This pressure-volume loop was taken from Patient 2, who suffered from acute respiratory distress syndrome, chronic pleural effusions, and chronic pleural thickening. The severe rightward shift in the slope reflects the low compliance of the chest wall. P_{ES} = esophageal pressure.

stant across treatments. This allowed all patients to consistently trigger breaths during the study period. V_T was set at the level prescribed for patient care. During PCV, the prescribed V_T usually was achieved by setting the pressure control level to the plateau pressure for the same V_T during VCV. By setting T_I at 1.0 second and maintaining a constant V_T, mean ventilator $\dot{V}_I$ was constant for each mode. Fractional concentration of inspired oxygen and PEEP were kept at the level prescribed by the managing physician. No patient care activities took place during the study period.

Measurements

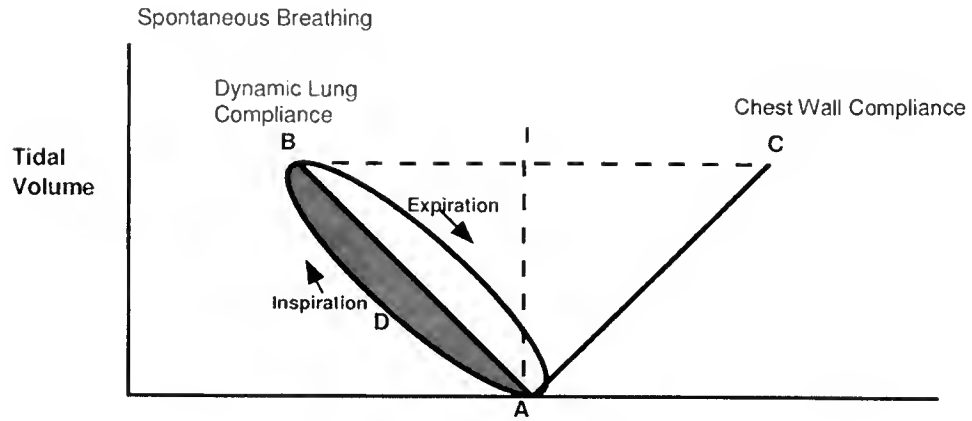
Patient and ventilator variables were measured with a pulmonary mechanics monitor incorporating a Campbell diagram (BICORE CP-100, Thermo Respiratory Group, Palm Springs, California). The precision and accuracy of this monitor has been previously validated.[15,16] The monitor and transducers were calibrated prior to each study. The Var-Flex (Thermo Respiratory Group, Palm Springs, California) airway pressure/flow transducer was placed at the patient Y adapter so that all reported volumes excluded compressible circuit volume. Data collection included f, inspired V_T, peak ventilator $\dot{V}_I$, intrinsic PEEP ($PEEP_I$), the fraction of T_I to total respiratory cycle time (ie, the duty cycle, calculated as T_I/T_{TOT}), and pressure-time product (PTP). Central respiratory drive was measured as the esophageal pressure at 100 milliseconds ($P_{0.1}$) after the onset of effort. Campbell diagram software was used to measure ΔP_{ES} and patient WOB in joules per liter (Fig. 2).[15] ΔP_{ES} was measured as the change in esophageal pressure from the X intercept of the chest wall compliance line from the Campbell diagram to the most negative point on the esophageal pressure-volume curve. $PEEP_I$ was measured as the difference in esophageal pressure between the X intercepts of the chest wall line (end-expiratory plateau) and lung compliance line (onset of inspiratory flow) from the Campbell diagram, minus the trigger sensitivity level measured at the airway (the lowest airway pressure change from baseline at the onset of flow). The mechanics monitor calculated PTP using the method described by Sassoon et al[17] as the integral of the negative change in P_{ES} over T_I, taking into account C_{CW}. However, the monitor is programmed to use a normal chest wall compliance value (200 mL/cm H_2O) in order to calculate PTP. Therefore, reported calculated values of PTP probably underestimate the true PTP as C_{CW} is often abnormal during ALI and ARDS.[18] The lung injury score was calculated using the method described by Murray et al.[19]

Fifteen to 20 assisted breaths were selected for analysis (Figs. 2–4). The criterion used for breath selection was a patient-triggered breath in which the P_{ES} remained below baseline after the onset of volume change. In one case (patient 5), respiratory drive was unstable, making it difficult to distinguish between well synchronized assisted ventilation and diminished drive. Therefore, we excluded all breaths from analysis when the WOB markedly fellbelow normal (< 0.08 J/L) in either mode. Dyssynchronous breaths associated with coughing and gross agitation, likewise, were excluded.

Calculations

The following formulas were used for calculation of the derived variables:

$$\dot{V}_E = V_T \times \text{total f (L/min)} \quad (1)$$



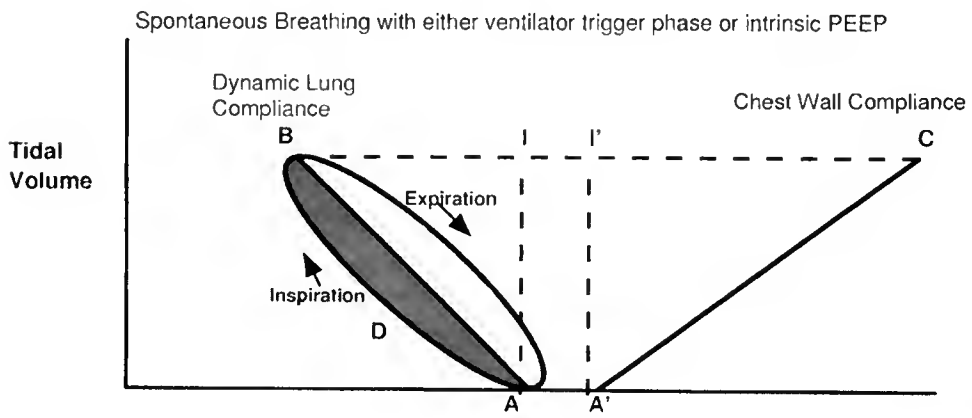


Fig. 2. A schematic drawing of a Campbell diagram in which line AB represents the lung compliance constructed dynamically during spontaneous breathing. Line AC represents the chest wall compliance constructed during passive ventilation and fitted to the end-expiratory pressure. The ellipse ADBA is the esophageal (or pleural) pressure-volume loop. The shaded area of the ellipse represents the flow-resistive work of breathing (WOB) performed by the patient. The area enclosed by ABCA represents the elastic WOB performed on the lungs and chest wall. Line AI bisects the elastic WOB so that area ABIA represents the elastic work performed on the lungs, whereas area AICA represents the elastic work performed on the chest wall. The unshaded portion of the ellipse represents passive expiration. Expiratory work is provided by the energy released during elastic recoil of the lungs to functional residual capacity. Spontaneous breathing through a ventilator's demand-flow system causes a trigger-phase so that a separation occurs between the lung and chest wall compliance lines. This signifies that the onset of effort (Point A') does not correspond to the onset of inspiratory flow (Point A). This separation between effort and flow also occurs in the presence of intrinsic positive end-expiratory pressure ($PEEP_I$). The increase in area ABCA'A is caused by rectangle AII'A', which represents the increased elastic work imposed by the trigger function of the ventilator and/or the presence of $PEEP_I$.

Respiratory muscle power in joules/minute

$$(\dot{W}) = \dot{V}_E \text{ (L/min)} \times \text{WOB (J/L)} \quad (2)$$

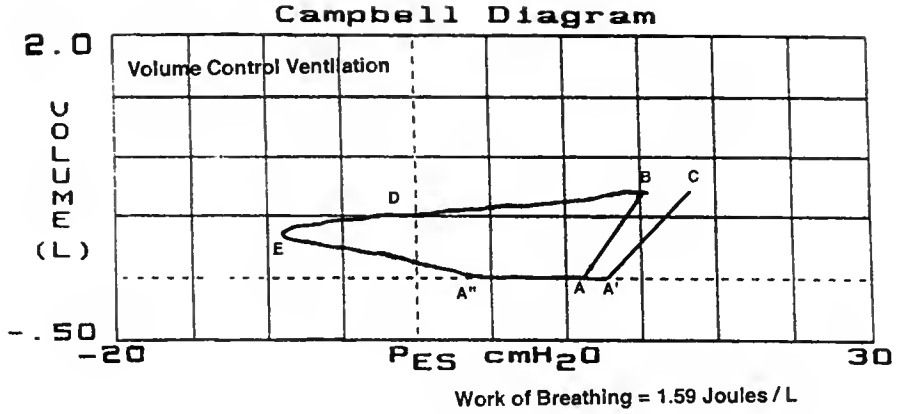
Spontaneous inspiratory time

$$= \text{spontaneous } f \times T_I/T_{TOT} \text{ (s)} \quad (3)$$

$$\text{Pressure time index (PTI)} = \Delta P_{ES}/MIP \times T_I/T_{TOT} \quad (4)$$

Data Analysis

Descriptive statistics are reported as means and standard deviations. The small sample size and non-normal data dis-



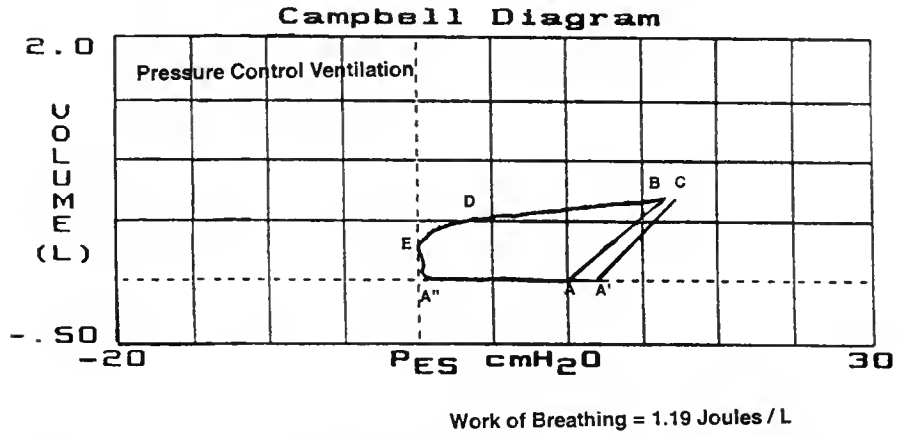


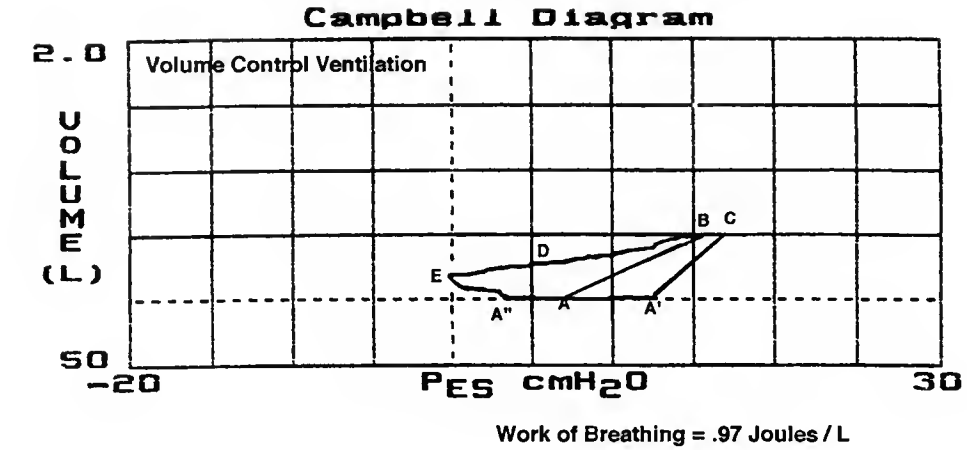
Fig. 3. Campbell diagram peak esophageal pressure vs tidal volume (P_{ES}-V_T) loops comparing high levels of assisted work of breathing (WOB) between volume control ventilation (VCV) and pressure control ventilation (PCV). The morphology of the pressure-volume loop is altered by the effect of positive pressure ventilation. A, B, C, D, and A' are as defined in the legend for Figure 2. Point A" is added to denote the post-trigger drop in P_{ES}, which represents the amount of effort exerted by the patient until there is sufficient flow delivery to cause a change in volume. Point E is added to denote peak negative P_{ES}, and the horizontal distance between A' and E represents peak esophageal pressure (ΔP_{ES}). The horizontal distance AE represents the portion of the ΔP_{ES} associated with the post-trigger phase. The differences in the magnitude of AE between VCV and PCV represents the effects of each flow pattern on the resistive WOB. The loops were matched for $\dot{V}_I$ and approximate agreement in baseline pressure (Point A'). The dynamic lung compliance line AB is not an accurate representation of lung compliance because of the contribution of positive airway pressure during the breath.

tribution required inferential statistical analyses using a Wilcoxon signed-rank test for paired comparisons between modes. Spearman's rank-order test was used to assess correlation (rho). Results were considered significant if $p < 0.05$. Data were analyzed using Stat View (Abacus Concepts, Berkeley, California).

Results

All 18 patients met either ALI or ARDS criteria at the time of study enrollment. One patient, recovering from ALI, had an improvement in his ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}) to > 300 after enrollment. The mean Murray[19] lung injury score revealed a moderate degree of damage (Table 1). Only 7 patients had scores indicative of severe injury (≥ 2.5) at the time of study.[19] The mean PEEP level was 6.7 ± 2.8 cm H_2O and the mean F_{IO_2} was 0.46 ± 0.08. Respiratory system compliance was in the range reported for ARDS (see Table 1).[7]

PCV was associated with significantly lower patient WOB, ΔP_{ES}, and $\dot{V}_I$ than was VCV (Table 2). PTP was



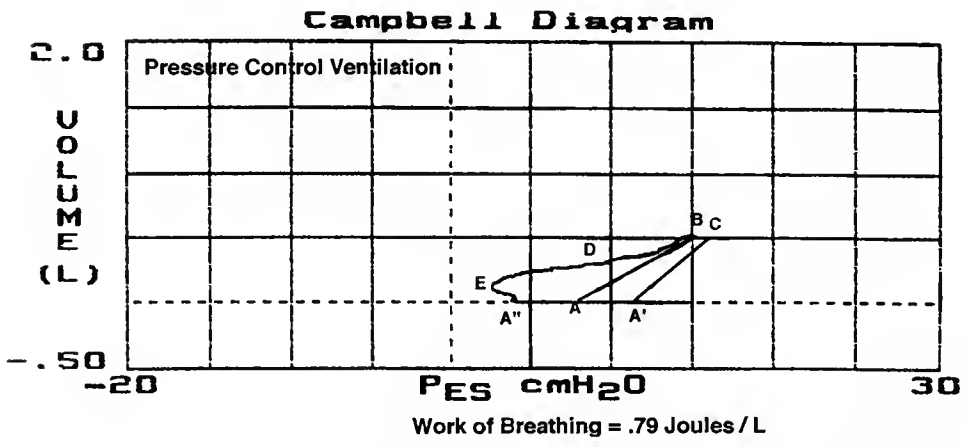


Fig. 4. Campbell diagram peak esophageal pressure vs tidal volume (P_{ES}-V_T) loops comparing moderate levels of assisted work of breathing (WOB) between volume control ventilation (VCV) and pressure control ventilation (PCV). The characteristic differences in the contour of the pressure-volume loops seen between VCV and PCV at high levels of work and effort also are present at moderate levels of work and effort. A, B, C, D, E, A′ and A″ are as defined in the legends for Figures 2 and 3.

lower during PCV, but the difference was not statistically significant. Central respiratory drive, or $P_{0.1}$, was the same between modes and was in the range reported for ARDS patients at 50% of total ventilator support.[4,20] Ventilator conditions were successfully maintained at constant levels between ventilator treatments (Table 3). V_T, total f, mean ventilator $\dot{V}_I$, and $\dot{V}_E$ were not different between assisted VCV and PCV. S_{pO_2} was monitored continuously and S_{pO_2} values were always $\geq$ 95% during data collection, in each mode. The pressure control level needed to maintain V_T was greater than the plateau pressure measured during VCV (32.7 $\pm$ 6.4 cm H_20 vs 29.7 $\pm$ 7.6 cm H_2O, respectively). Peak ventilator $\dot{V}_I$ was significantly greater during PCV. However, mean ventilator $\dot{V}_I$ was low relative to general practice (41 L/min vs 60 L/min).[21] Mean V_T as a function of body size was 10.0 $\pm$ 2.5 mL/kg.

We were able to measure inspiratory muscle strength at the beginning of the study in all patients. The MIP revealed a moderately strong inspiratory muscle force reserve (see Table 1). Our results were similar to those reported by Marini et al for critically ill patients, using the same measurement technique.[13] In addition, we were able to assess the breathing pattern and WOB during brief periods of spontaneous ventilation in 16 of 18 patients at the end of the study (Table 4). The peak $\dot{V}_I$, mean $\dot{V}_I$, and V_T that could be generated during spontaneous breathing was lower than the respective values measured during VCV and PCV. Mean patient T_I was close to ventilator T_I (0.9 s vs 1.0 s,

Table 1. Patient Demographics at Entrance into Study

Case	Sex	Age	Diagnosis	C_{RS} (mL/cm H_2O)	C_{CW} (mL/cm H_2O)	PEEP (cm H_2O)	LIS	MIP (cm H_2O)
1	M	47	Pneumonia, ALI	50	143	5	0.75	14
2	F	57	ARDS, ESLD, chronic pleural effusion/ pleural thickening	16	46	8	2.75	26
3	M	19	Smoke inhalation, resolving ARDS	28	109	5	1.75	75
4	F	33	Hypovolemic shock, ascites, ARDS	21	90	15	2.5	39
5	F	65	Blunt chest trauma, pulmonary contusion, pneumonia, ALI	41	146	5	1.75	44
6	M	44	Abdominal abscesses, pleural effusion, resolving ALI	45	175	5	1.00	53
7	F	49	Blunt chest trauma, flail chest, ALI, aspiration pneumonia	40	256	7	2.00	27
8	M	38	Blunt chest trauma, pulmonary contusion, flail chest, ALI	50	262	5	1.50	32
9	M	70	Sepsis, ALI, COPD	39	*	5	1.33	71
10	M	61	Pneumonia, ALI, sepsis	33	129	5	2.67	56
11	F	33	Resolving ARDS, sepsis	25	205	5	1.33	63
12	F	39	Pneumonia, ARDS, ESLD	35	200	5	1.75	45
13	M	38	Blunt chest trauma, ARDS	23	*	5	2.50	60
14	F	51	Pancreatitis, ESLD, sepsis, ARDS	24	126	10	3.00	57
15	M	66	Pneumonia, sepsis, ALI	38	131	5	1.50	23
16	M	39	Multiple stab wounds to chest/abdomen, ARDS	21	39	10	2.75	23
17	M	76	Pneumonia, ARDS, COPD	28	*	5	1.75	29
18	F	42	Necrotizing faciitis, ARDS, sepsis	26	164	10	2.50	57
Mean ± SD		48.2 ± 15.0		32 ± 11	137 ± 70	6.7 ± 2.8	1.95 ± .66	44.1 ± 18.3

C_{RS} = respiratory system compliance. C_{CW} = chest wall compliance. PEEP = positive end-expiratory pressure. LIS = lung injury score (ARDS Score).[19] MIP = maximal inspiratory pressure (esophageal). ALI = acute lung injury. ARDS = acute respiratory distress syndrome. ESLD = end-stage liver disease. COPD = chronic obstructive pulmonary disease. * = data unobtainable.

Table 2. Patient Variables Between VCV and PCV During Assisted Ventilation

Variable	VCV	PCV
$P_{0.1}$ (cm H_2O)	4.17 ± 5.14	4.05 ± 4.38
ΔP_{ES} (cm H_2O)	9.14 ± 5.48	7.45 ± 5.65*
PTP (cm $H_2O \cdot s \cdot min$)	95.7 ± 59.3	83.2 ± 53.8
WOB (J/L)	0.70 ± 0.58	0.59 ± 0.42†
$\dot{W}$ (J/min)	8.22 ± 4.42	6.92 ± 4.36*

Values are reported as mean ± standard deviation.
VCV = volume control ventilation.
PCV = pressure control ventilation.
$P_{0.1}$ = esophageal pressure in the first 100 miliseconds.
ΔP_{ES} = peak esophageal pressure.
PTP = pressure-time product.
WOB = work of breathing.
$\dot{W}$ = power output of the inspiratory muscles.
*$p < 0.01$.
†$p < 0.05$.

Table 3. Ventilator Variables Between VCV and PCV During Assisted Ventilation

Variable	VCV	PCV
V_T (mL/breath)	674 ± 88	685 ± 97
Total f (breaths/min)	18.2 ± 3.3	18.1 ± 3.3
Peak $\dot{V}_I$ (L/min)	43.8 ± 8.4	103.2 ± 22.8*
Peak P_{AW}	40.7 ± 8.4	32.7 ± 6.4†
Mean $\dot{V}_I$ (L/min)	40.7 ± 5.3	41.1 ± 5.8
$\dot{V}_E$ (L/min)	12.4 ± 2.9	12.5 ± 3.3

Values are reported as mean ± standard deviation.
VCV = volume control ventilation.
PCV = pressure control ventilation.
V_T = tidal volume.
f = respiratory frequency.
$\dot{V}_I$ = inspiratory flow rate.
P_{AW} = airway pressure.
$\dot{V}_E$ = minute ventilation.
*$p < 0.001$.
†$p < 0.05$.

respectively). Measurements of WOB, $\dot{V}_I$, pressure-time index, PTP, and the rapid-shallow breathing index (f/V_T) made during spontaneous breathing exceeded the levels thought to induce (or indicate) respiratory muscle fatigue and weaning intolerance. Therefore, our sample generally appeared to be ventilator-dependent.

A confounding variable ($PEEP_I$) was detected in 4 of 18 patients. In 3 cases (patients 3, 4, and 17) $PEEP_I$ was slightly higher during PCV than during VCV (3.03 cm H_2O vs 1.03 cm H_2O). However, WOB was less during PCV than VCV (0.73 J/L vs 0.82 J/L). All three of these

Table 4. Spontaneous Breathing Variables Measured in 16 Patients

Variable	Mean ± SD	Weaning Threshold
V_T (mL/breath)[23]	300 ± 120	≥ 400
f (breaths/min)[24]	30.1 ± 10.4	< 35
$\dot{V}_E$ (L/min)[25]	9.00 ± 4.99	< 10.0
f/V_T[26]	115.9 ± 58.9	< 105
Peak $\dot{V}_I$ (L/min)	33.0 ± 10.8	
Mean $\dot{V}_I$ (L/min)	20 ± 8.1	
ΔP_{ES} (cm H_2O)	14.2 ± 5.9	
$\Delta P_{ES}/MIP$[27]	0.39 ± 0.16	< 0.40
WOB (J/L)[28]	1.40 ± 0.77	< 0.95
$\dot{W}$ (J/min)[29]	12.37 ± 9.93	< 10.0
T_I/T_{TOT}	0.41 ± 0.09	
T_I	0.89 ± 0.33	
PTI[30]	0.16 ± 0.06	0.15
PTP (cm $H_2O \cdot s \cdot min$)[17]	279.5 ± 127.3	< 150

V_T = tidal volume.
f = respiratory rate.
$\dot{V}_E$ = minute ventilation.
f/V_T = rapid shallow breathing index.
$\dot{V}_I$ = inspiratory flow rate.
ΔPES = peak esophageal pressure.
$\Delta P_{ES}/MIP$ = ΔPES as a fraction of maximal inspiratory esophageal pressure.
WOB = inspiratory work per liter of ventilation.
$\dot{W}$ = inspiratory muscle power output.
T_I/T_{TOT} = inspiratory time fraction.
T_I = inspiratory time.
PTI = pressure-time index.
PTP = pressure-time product.

Table 5. Differences in Dependent Measures Between VCV and PCV During Assisted Ventilation After Excluding Patients with $PEEP_I$

Variable	VCV	PCV
$P_{0.1}$ (cm H_2O)	4.54 ± 5.81	4.09 ± 4.94
ΔP_{ES} (cm H_2O)	6.78 ± 5.75	5.63 ± 5.84
PTP (cm $H_2O \cdot s \cdot min$)	90.45 ± 62.21	66.2 ± 39.70*
WOB (J/L)	0.56 ± 0.56	0.49 ± 0.34†
$\dot{W}$ (J/min)	7.23 ± 4.45	6.10 ± 4.61‡
V_T (mL/breath)	680 ± 80	690 ± 90
$\dot{V}_E$ (L/min)	12.44 ± 2.46	12.26 ± 2.94

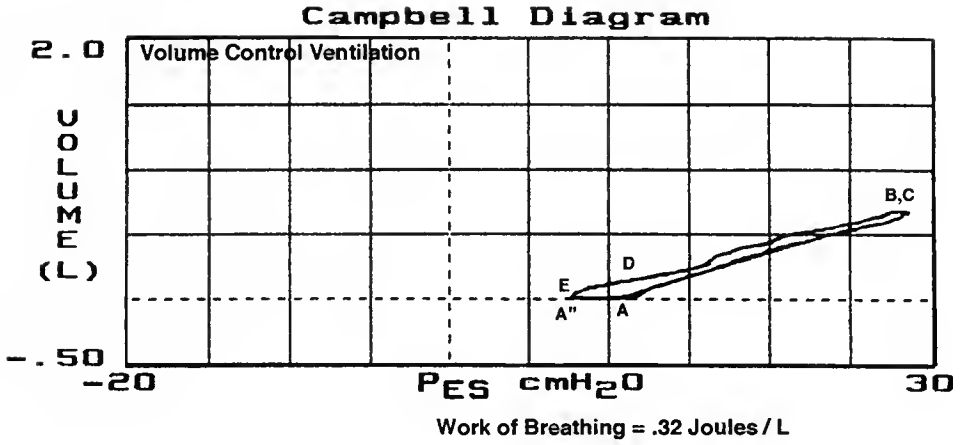
Values are reported as mean ± standard deviation.
VCV = volume control ventilation.
PCV = pressure control ventilation.
$P_{0.1}$ = esophageal pressure in the first 100 milliseconds.
ΔPES = peak esophageal pressure.
PTP = pressure-time product.
WOB = work of breathing.
$\dot{W}$ = power output of the inspiratory muscles.
V_T = tidal volume.
$\dot{V}_E$ = minute ventilation.
*$p < 0.01$.
†$p < 0.05$.
‡$p < 0.001$.

patients had an underlying component of chronic obstructive pulmonary disease or asthma. Expiratory activity was minimal in either mode. In the remaining case (patient 10) $PEEP_I$ varied greatly between modes (5.1 cm H_2O vs 12.3 cm H_2O for PCV and VCV, respectively) as did WOB (1.68 J/L and 2.28 J/L for PCV and VCV, respectively). Active expiratory effort was prominent during VCV. We suspected that $PEEP_I$ was attributable to mild dynamic hyperinflation in the first 3 patients and active expiration in patient 10.[22,31] When the data were reanalyzed after excluding all cases with $PEEP_I$, PTP was significantly lower during PCV ($p < 0.05$) (Table 5). All other significant results of dependent variables from the main data pool remained significant during reanalysis. V_T, total f, $\dot{V}_I$, and $P_{0.1}$ were not different between modes when all cases with $PEEP_I$ were removed.

Discussion

The main finding of our study was that PCV was more effective than VCV in reducing patient WOB, ΔP_{ES}, and $\dot{W}_I$ during assisted ventilation at a constant mean ventilator $\dot{V}_I$. When $PEEP_I$ was controlled for in the analysis, PTP also was significantly less with PCV. Both PTP and $\dot{V}_I$ are mechanical correlates of respiratory muscle oxygen consumption.[32] We believe that the attributes of PCV (a high peak ventilator $\dot{V}_I$, demand-responsive changes in peak ventilator $\dot{V}_I$, and a sustained high $\dot{V}_I$ when patient effort is prolonged) were responsible for the lower patient WOB with PCV, relative to VCV. The most important factor probably was the peak ventilator $\dot{V}_I$, which was significantly higher during PCV than during VCV. The demand-responsive changes in peak ventilator $\dot{V}_I$ and a sustained high $\dot{V}_I$ were assessed by the variability in both peak ventilator $\dot{V}_I$ and V_T, respectively. We calculated the standard deviation in the breaths used for analysis in individual subjects in each mode. We found that the mean standard deviation for peak ventilator $\dot{V}_I$ was over two and one half times higher during PCV than during VCV (9.6 L/min and 3.6 L/min, respectively). In each patient, V_T variability was not different between the modes. Therefore, the ability of PCV to vary peak ventilator $\dot{V}_I$ on a breath-to-breath basis (and not variability in V_T size) also may have contributed to reducing patient WOB during PCV.

We hypothesized that PCV would diminish both the elastic and resistive components of work. The Campbell diagram is used to assess the total mechanical WOB by accounting for the elastic load contributed by the chest wall.[33] Although the Campbell diagram also can be used to differentiate elastic from resistive work during spontaneous breathing[33] and weaning,[14,34] its use is not valid during high levels of assisted ventilation.[14] However, inspection of the P_{ES}-V_T curves generated during assisted ventilation may provide some insight into the nature of the total work



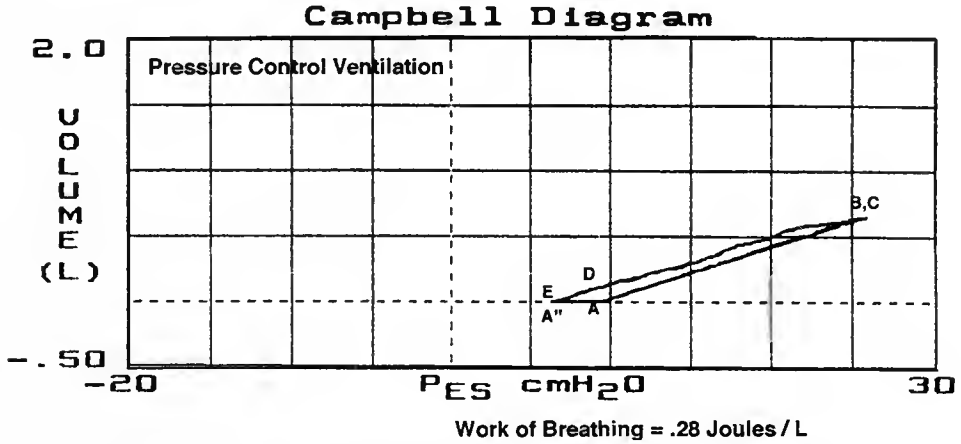


Fig. 5. Campbell diagram peak esophageal pressure vs tidal volume (P_{ES}-V_T) loops comparing low levels of assisted work of breathing (WOB) between volume control ventilation (VCV) and pressure control ventilation (PCV). The loops were matched for V_T and approximate agreement in baseline pressure. A, B, C, D, E, A′ and A″ are as defined in the legends for Figures 2 and 3. The contour of the pressure-volume loops are almost indistinguishable between VCV and PCV. These tracings suggest the equivalence in WOB reduction between modes at low levels of effort.

performed by patients in each mode. The P_{ES}-V_T curves from assisted VCV and PCV (Figs. 3–5) were characteristic of the differences in waveform morphology found across the sample between modes. At both high and moderate levels of patient work, P_{ES} continued to move in a negative direction despite constant volume change during VCV (see Figs. 3 and 4). We interpreted this as indicating the presence of imposed resistive WOB. Furthermore, P_{ES} often remained below the end-expiratory (baseline) pressure at end-inspiration. This indicated to us that the volume control breath did not reduce the elastic WOB associated with lung inflation and chest wall displacement. During PCV, P_{ES} tended to change in a positive direction at a lower volume (see Fig. 3). This tendency supports the concept that PCV's rapid ventilator $\dot{V}_I$ and V_T delivery may exceed the contractile velocity of the inspiratory muscles, thereby arresting the drop in muscle pressure and thus reducing both the resistive and elastic WOB. At low levels of patient effort the P_{ES}-V_T curves were almost indistinguishable, with minimal differences in work (see Fig. 5). This suggests that patient WOB is not different between modes at low effort.

To our knowledge, only Cinnella et al[35] have directly compared patient WOB between assisted VCV and PCV. They reported no differences in patient WOB at 12 mL/kg V_T but significantly less work during PCV at 8 mL/kg V_T. As in our study, Cinnella et al kept V_T and mean ventilator $\dot{V}_I$ constant between modes. In our study V_T was kept at the level used for clinical management (10.0 ± 2.5 mL/kg). We found no relationship between V_T size and patient WOB in either mode (rho = 0.09). We believe the differences in results between studies are explained by peak ventilator $\dot{V}_I$ performance during PCV. Peak ventilator $\dot{V}_I$ during VCV was comparable to our study (43.8 L/min vs

43.2 L/min, respectively) and the pre-set T_I in each mode was the same as in our study (1 s). However, the peak ventilator $\dot{V}_I$ during PCV reported by Cinnella et al[35] was much lower than in our study (54.6 L/min vs 103.2 L/min, respectively). The small differences in peak ventilator $\dot{V}_I$ between modes reported by Cinnella et al[35] were probably due to the fact that many of their subjects had severe obstructive lung disease with high inspiratory resistance. High inspiratory resistance decreases peak ventilator $\dot{V}_I$ in PCV because less flow is needed to achieve the target airway pressure.[36] In addition, high inspiratory resistance during PCV causes ventilator $\dot{V}_I$ to taper off slowly (because of the slower rate-rise in alveolar pressure)[36] so that the flow waveform was transformed in the Cinnella study from an exponential decelerating to a quasirectangular pattern (L Brochard, Henri Mondor Hospital at the University of Paris, 2000, personal communication). The differences in WOB at 8 mL/kg V_T for the entire sample were probably due to the larger differences in peak ventilator $\dot{V}_I$ between VCV and PCV in the subset of patients without obstructive lung disease (45 L/min vs 67 L/min, respectively).[35]

Two related studies compared the effects of variable versus constant flow wave forms on WOB. Haas et al[37] compared VCV to volume-assured pressure support ventilation (VAPS). During VAPS, peak ventilator $\dot{V}_I$ was higher (63 L/min) than during VCV (54 L/min), although mean ventilator $\dot{V}_I$ appears to have been the same as in our study. The differences in patient WOB between VAPS and VCV was similar to ours (0.43 J/L vs 0.52 J/L, respectively). However, VAPS does not function the same as PCV. In VAPS both peak airway pressure and minimum V_T are set so that the ventilator controls $\dot{V}_I$ according to two algorithms. The ventilator $\dot{V}_I$ wave form of VAPS was a hybrid resembling a half-sine wave during early inspiration and a constant flow pattern in the later part of inspiration.[37]

MacIntyre et al[38] compared the effectiveness of treating patient-ventilator flow-dyssynchrony either by increasing ventilator $\dot{V}_I$ during VCV (by shortening T_I) or by adding a "pressure-limiting feature" to the breathing pattern. This latter approach appeared to alter peak ventilator $\dot{V}_I$ by incorporating a decelerating flow pattern. PTP (in cm H_2O) rather than WOB (in J/L) was measured. Although there was an improvement in PTP during the pressure-limited breath delivery, the difference was not statistically significant.[38] Comparing our study to that of MacIntyre et al[38] is difficult. In their study, differences in V_T, T_I, and f occurred between treatments and the resulting differences in both peak and mean ventilator $\dot{V}_I$ across treatments were not reported.

MacIntyre et al[38] treated ventilator $\dot{V}_I$ dyssynchrony with VCV by reducing T_I to approximately 0.5 second (compared to approximately 1 s during the pressure-limited breaths) in order to achieve comparable levels of work reduction.[38] Depending on ventilator design, shortening T_I may have a significant clinical impact. The Siemens 900C cannot set T_I independently of the control f. In addition, V_T is determined by both the control f and the pre-set $\dot{V}_E$. Therefore, in ventilators designed like the Siemens 900C, treating ventilator $\dot{V}_I$ dyssynchrony by shortening T_I (at a constant V_T) requires a higher f and pre-set $\dot{V}_E$. This may exceed both the f and $\dot{V}_E$ requirements of the patient, resulting in hypocapnia and complete suppression of spontaneous effort. Also, preliminary evidence suggests that using a high mean ventilator $\dot{V}_I$ may inadvertently increase respiratory drive and may cause dyssynchrony between the patient and ventilator.[39]

Our study did not address whether raising peak ventilator $\dot{V}_I$ during VCV to equal that achieved during PCV accomplishes the same reduction in WOB. To approach the peak ventilator $\dot{V}_I$ achieved during PCV (103.2 L/min) with VCV would have required a pre-set $\dot{V}_I$ that exceeded patient demand. It is of interest to note that our study, as well as the studies by Cinella et al[35] and Hass et al,[37] all set VCV with a mean ventilator $\dot{V}_I$ significantly less than 60 L/min. However, when MacIntyre et al[38] used a mean ventilator $\dot{V}_I$ of 76 L/min to treat $\dot{V}_I$ dyssynchrony, PTP was not significantly different. In fact, PTP was higher during VCV than during pressure-limited breaths at a more appropriate T_I.[38] Several recent studies have all reported that high ventilator $\dot{V}_I$ delivered with a rectangular wave causes respiratory center excitation manifested by increased *f*.[39–42] Bshouty and Georgopoulos[43] found that high mean ventilator $\dot{V}_I$, rather than peak ventilator $\dot{V}_I$, may be responsible for the respiratory center excitation. Therefore, a high peak ventilator $\dot{V}_I$ with VCV may lessen patient WOB, but at a certain point this may be countered by the effects of high mean ventilator $\dot{V}_I$ on respiratory drive. In addition, our study did not address the issue of whether using VCV with a decelerating flow wave form would have produced the same results. We think it is reasonable to predict that VCV with a decelerating flow wave form would reduce WOB more than VCV with a constant or rectangular flow wave form. However, peak ventilator $\dot{V}_I$ would remain fixed and would not necessarily be adequate in situations where patient flow demand was both vigorous and fluctuating.

Conclusions

In conclusion, PCV may help in the management of critically ill patients with ALI and ARDS by reducing patient WOB, $\dot{W}$, and PTP. We attributed these reductions to the characteristic high, variable ventilator $\dot{V}_I$ and faster V_T delivery of PCV. In addition, in ventilators where T_I cannot be set independently of control f, PCV allows for

better flow matching without the necessity of setting backup f and $\dot{V}_E$ in excess of patient demand.

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Nitric Oxide Delivery During High-Frequency Oscillatory Ventilation

Yuji Fujino MD, Robert M Kacmarek PhD RRT FAARC, and Dean R Hess PhD RRT FAARC

BACKGROUND: Inhaled nitric oxide (NO) is used increasingly in the care of infants with hypoxemic respiratory failure and is frequently combined with high-frequency oscillation (HFO). The aim of this study was to evaluate delivery of NO during HFO using titration into the ventilator circuit or using the INOvent Delivery System. METHODS. NO was delivered into the HFO circuit at three sites (pre-humidifier, post-humidifier, and after the bellows) by continuous titration using a rotameter. The target NO concentration ([NO]) was initially adjusted using a rapid-response chemiluminescence NO analyzer without oscillation at 5, 10, and 20 parts per million (ppm). During the study, gas was sampled 5 cm from the bellows (proximal), 35 cm from the bellows (middle), and at the distal end of the circuit (distal). The ventilator was set at frequencies of 5, 10, and 15 Hz, mean airway pressures of 15, 20, and 25 cm H_2O, and amplitudes of 20, 30, and 40 cm H_2O. Soft and hard circuits were evaluated. The fraction of inspired oxygen was 0.90, the inspiratory time fraction was 33%, and the bias flow was 20 L/min throughout the study. An INOvent Delivery System was also evaluated with the same HFO settings. RESULTS. The fluctuation of [NO] was minimal with continuous titration pre-humidifier at all HFO settings. [NO] fluctuated with titration post-humidifier and after the bellows, especially at the proximal sampling site. At the lung model, however, fluctuation of [NO] was always < 1.5 ppm and usually < 1 ppm. Delivered [NO] was lower than target [NO] with injection after the bellows (> 5%). The soft circuit showed better mixing of NO than the hard circuit. The INOvent Delivery System delivered a stable and accurate [NO] at all settings. [NO_2] was < 1 ppm at all settings. CONCLUSIONS. Mixing of NO during HFO was acceptable at all the injection sites evaluated, although injection pre-humidifier was preferable because of small fluctuations of [NO]. The INOvent Delivery System was simple to use and delivered an accurate and precise [NO] during HFO. *Key words: inhaled nitric oxide, inhaled nitrogen dioxide, high-frequency oscillation, mechanical ventilation, nitric oxide delivery system, nitric oxide, nitrogen dioxide, chemiluminescence, persistent pulmonary hypertension of the newborn.* [Respir Care 2000;45(9):1097–1104]

Background

Inhaled nitric oxide (NO) is used to reduce pulmonary artery pressure and increase arterial oxygenation in newborn patients with pulmonary hypertension and hypoxemia.[1,2] High-frequency oscillation (HFO) is also used in the treatment of these patients and has been reported to augment the beneficial effects of inhaled NO.[3–6] Inhaled NO has been administered during HFO by titration into the continuous gas flow.[7,8] It is important that the infused NO adequately mixes in the bias gas flow before delivery to the patient to assure a stable inspired NO concentration ([NO]). Inadequate mixing may result in imprecise NO delivery, with the potential for either toxicity or a clinically ineffective dose.

During conventional ventilation with an adult ventilator, continuous titration of NO into the ventilator circuit has been reported to produce [NO] fluctuations of nearly 10-fold.[9,10] Unless a rapid-response NO analyzer is used, this fluctuation is undetected.[11] There has been little study of NO delivery with HFO to determine whether [NO] fluctuates beyond the clinically acceptable range using the titration method and a rapid-response analyzer. More re-

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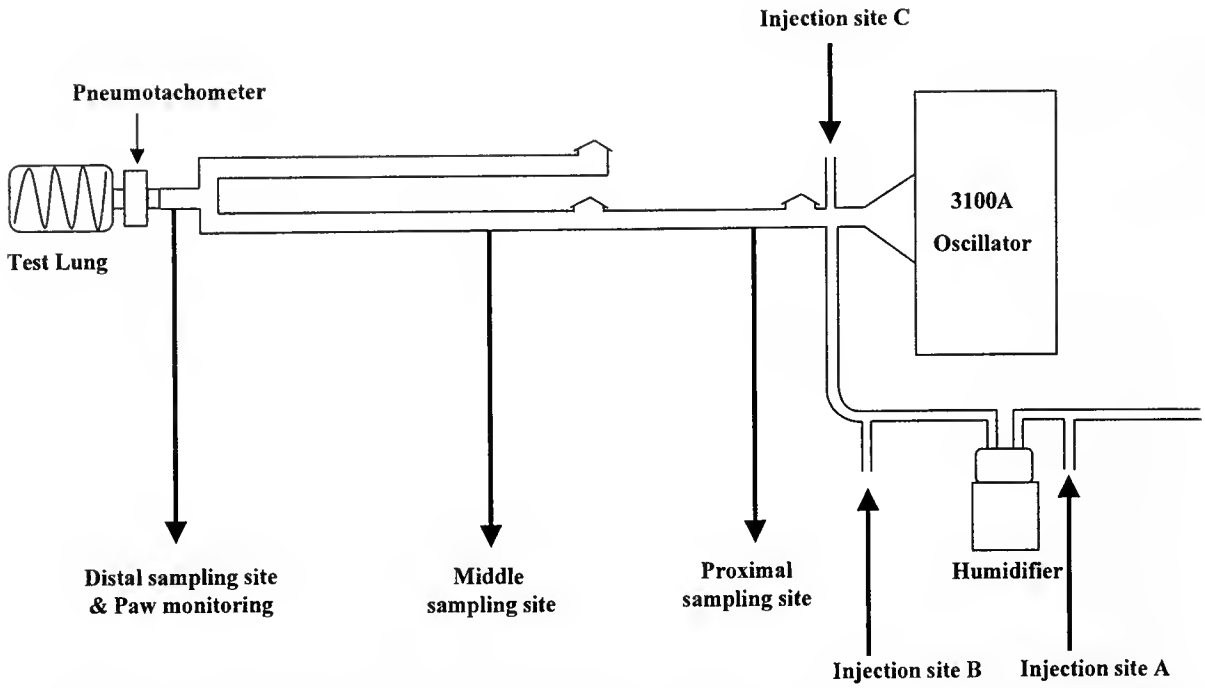

Fig. 1. Schematic diagram of the experimental setup. NO flow was introduced into the inspiratory limb of the circuit at three sites: before the humidifier (injection site A), distal to the humidifier (injection site B), distal to the oscillator bellows (injection site C). The gas sample for the chemiluminescence analyzer was obtained from 3 cm distal to the injection site C (proximal), 30 cm distal to the proximal site (middle), and at the temperature probe connection port close to the Y-piece (distal). Paw = airway pressure.

cently, a delivery system for inhaled NO, the INOvent Delivery System (INO Therapeutics, Clinton, New Jersey), has become available. Although the INOvent has been reported to deliver a stable [NO] during conventional adult ventilation,[12,13] its performance during HFO has not been reported.

The aim of this study was to evaluate the mixing of NO with titration into the HFO ventilator circuit, or with the use of the INOvent, with a fast-response NO analyzer. Our hypothesis was that NO can be safely administered during HFO using either direct titration into the circuit or with the use of the INOvent Delivery System.

Methods

Experimental Setup

NO was provided in tanks containing 800 parts per million (ppm) balanced in nitrogen (N_2) (INOmax, INO Therapeutics, Clinton, New Jersey). The NO flow (Timeter Classic 1000 or 200 oxygen flow meter, The Timeter Group, St Louis, Missouri) was introduced into the inspiratory limb of the circuit at 3 sites: before the humidifier (Conchatherm III, Hudson Respiratory Care, Temecula, California) (injection site A), distal to the humidifier (injection site B), and distal to the oscillator bellows (injection site C) (Fig. 1). NO delivery was also evaluated using the INOvent Delivery System at site A (before the humidifier) with a one-way valve at the outlet of the INOvent injector, as recommended by the manufacturer.

Gas for analysis by chemiluminescence was sampled 3 cm distal to injection site C (proximal), 30 cm distal to the proximal site (middle), and at the temperature probe port close to the Y-piece (distal). Airway pressure was measured at the distal sampling site. A pneumotachometer and gas sampling site for the electrochemical analyzer (INOvent) to measure NO_2 concentration were connected between the Y-piece and the test lung (copper wool-filled container with a compliance of 0.93 mL/cm H_2O).

Settings for High-Frequency Oscillation

Frequency was set at 5, 10, and 15 Hz. The inspiratory time (T_I) was set at 33% throughout. Mean airway pressure ($\bar{P}_{AW}$) was set at 15, 20, and 25 cm H_2O, and amplitude (P_{AMP}) was set at 20, 30, and 40 cm H_2O with a bias flow of 20 L/min. The fraction of inspired oxygen (F_{IO_2}) was set at 0.90. The pressure limit of the ventilator was set at maximum (45 cm H_2O). The humidifier in the circuit was not used and measurements were performed in a dry gas. Hard and soft type circuits provided by the manufacturer were evaluated.

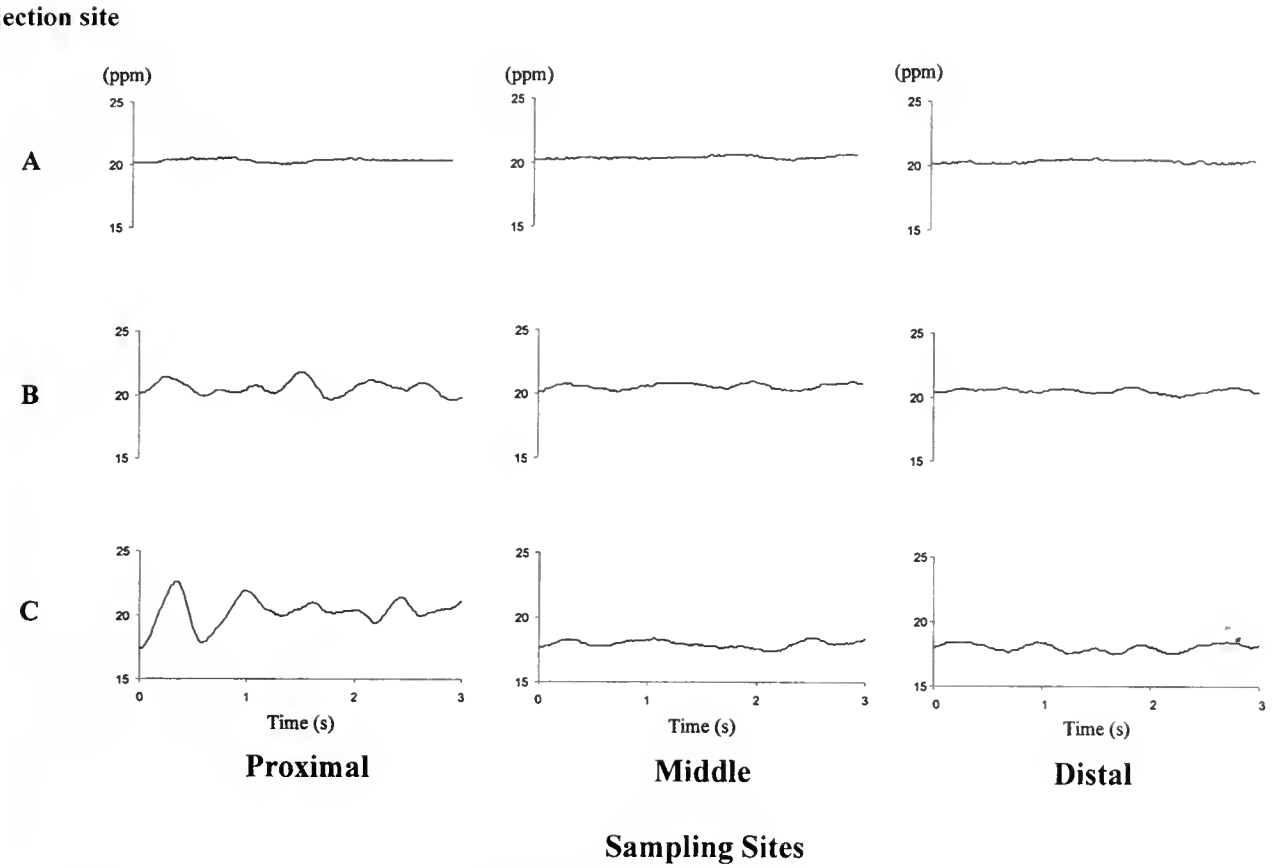

Fig. 2. Actual tracings of NO concentration ([NO]) in parts per million (ppm) with frequency 10 Hz, mean airway pressure ($\bar{P}_{AW}$) 25 cm H_2O, and amplitude (P_{AMP}) 20 cm H_2O at a target [NO] of 20 ppm with the hard circuit. When NO was injected at site A, [NO] was flat at all sampling sites. With B and C injection sites, [NO] fluctuation was prominent at the proximal sampling site, but still observed at the middle and distal sampling sites.

Experimental Protocol

The target [NO] (5, 10, and 20 ppm) was titrated without oscillation. After measurements of each $\bar{P}_{AW}$ and P_{AMP}, oscillation was interrupted and [NO] was measured again without oscillation. Each target [NO] was evaluated in this manner. For all reported measurements, the drift of [NO] before and after the each experimental condition was $< 5\%$ of target [NO].

Measurement and Calibration

A chemiluminescence analyzer (Sievers model NOA 280, Sievers Instruments, Boulder, Colorado) and the electrochemical analyzer incorporated into the INOvent were used. This chemiluminescence analyzer has a 95% response time of 220 ms for NO.[11] Both were calibrated with 26.6 ppm NO in N_2 (INO Therapeutics, Clinton, New Jersey). The INOvent was also calibrated with 13 ppm NO_2 (INO Therapeutics, Clinton, New Jersey) and 100% O_2. [NO] from the chemiluminescence analyzer only was used for purposes of this study. The airway pressure was measured using a pressure transducer (model 45–32-871 $\pm$ 100, Validyne, Northridge, California) calibrated with a water manometer. Gas flow was measured using a pneumotachometer (3700A, Hans Rudolph Inc, Kansas City, Missouri) calibrated with a precision rotameter. Tidal volume was integrated using the flow signal and confirmed with a 10 mL syringe. All signals were amplified (model 8805C, Hewlett Packard, Waltham, Massachusetts) and recorded at 500 Hz using an analog-to-digital conversion system (Windaq v1.36, Dataq Instruments, Akron, Ohio) and a personal computer. All devices were calibrated at the beginning of the experiment.

Data Analysis

Maximum, mean, and minimum [NO] over 5 seconds from the chemiluminescence analyzer were evaluated for the fluctuation of [NO]. The difference between maximum and minimum [NO] is reported as Δ[NO].

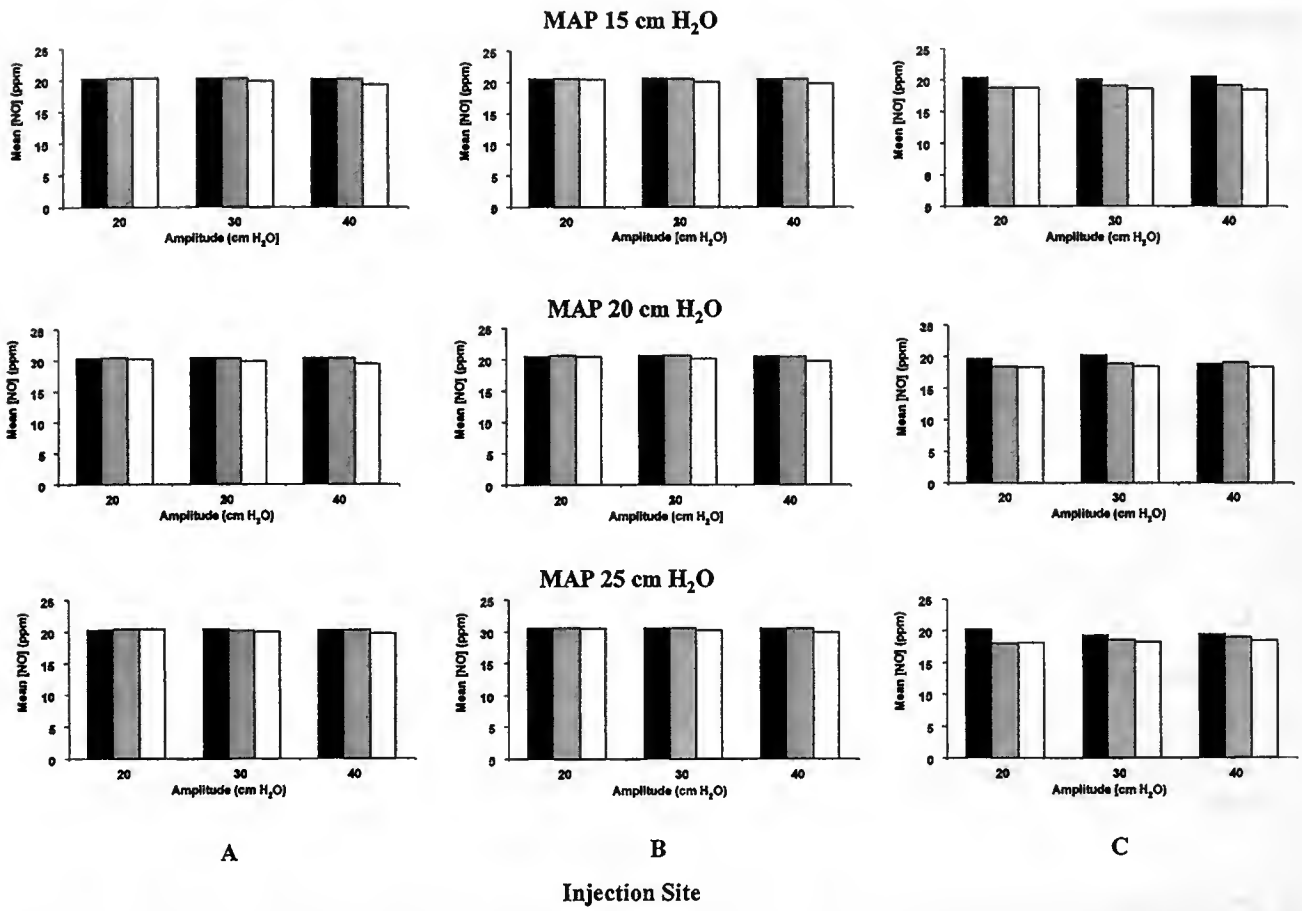

Fig. 3. Mean NO concentration ([NO]) in parts per million (ppm) with each mean airway pressure (MAP) and amplitude at different injection and sampling sites with the hard circuit. Data are only presented for target NO concentration ([NO]) of 20 ppm and frequency 10 Hz. Closed bars represent proximal sampling, gray bars represent middle sampling, and open bars represent distal sampling. A: pre-humidifier. B: post-humidifier. C: post-bellows.

Results

[NO] was stable at all sample sites when NO was injected before the humidifier (site A) (Fig. 2). When NO was injected at sites B or C, [NO] fluctuation was present at the proximal sampling site (Figs. 2–4). The fluctuation in [NO] was greatest at the proximal sampling site at all settings. At the lung model, [NO] fluctuation was always $<$ 1.5 ppm and usually $<$ 1 ppm. The greater the amplitude, the smaller the [NO] fluctuation at B and C injection sites (see Fig. 4). At site C (after the bellows), the measured [NO] at the middle and distal sampling sites was lower than target [NO] ($>$ 5%) with each $\bar{P}_{AW}$ and P_{AMP} (see Fig. 3). The greater the $\bar{P}_{AW}$, the greater the [NO] fluctuation at C injection site and proximal sampling site (see Fig. 4).

With the soft circuit, measured [NO] at the proximal sampling site was lower than the target [NO] ($>$ 5%). However, measured [NO] at the distal sampling site was virtually the same as the target [NO] (Fig. 5). With the INOvent, measured [NO] was nearly the same as the target [NO] during all settings and showed only small fluctuations (see Fig. 5).

With all settings, measured [NO_2] at the distal sampling site was $<$ 1 ppm. Measured [NO_2] showed a tendency to decrease at the higher P_{AMP} (Table 1).

Discussion

The major findings of this study are as follows.

(1) Nitric oxide was well mixed with the continuous gas flow at the proximal airway with all injection sites we evaluated.

(2) When nitric oxide is injected after the bellows of the oscillator with a hard circuit, the delivered [NO] may be lower than the target dose.

(3) The INOvent Delivery System accurately delivers the target [NO].[4] Measured [NO_2] was clinically acceptable at all oscillator settings.

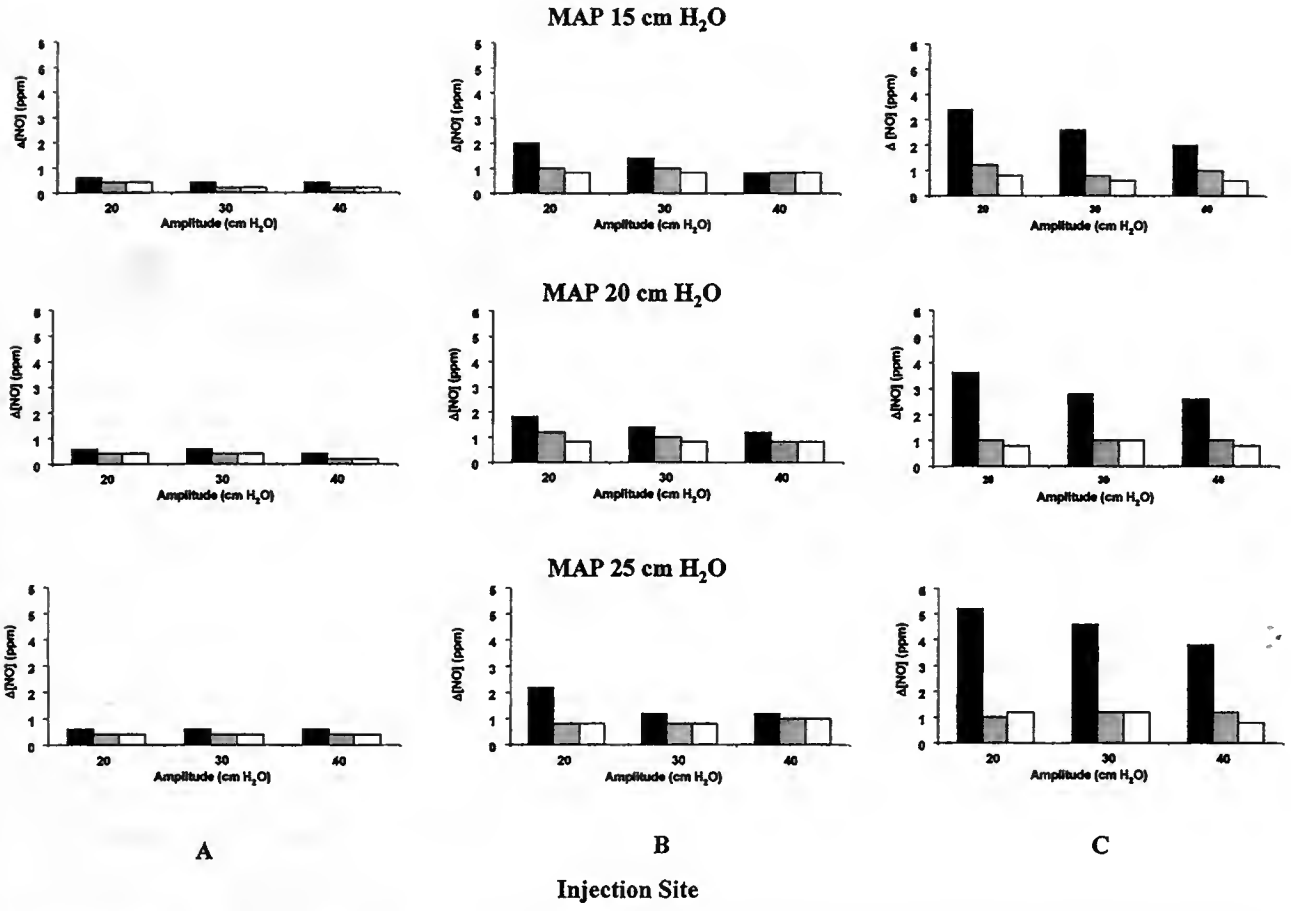

Fig. 4. Fluctuation of NO concentration (Δ[NO]) in parts per million (ppm) with each mean airway pressure (MAP) and amplitude at different injection and sampling sites with the hard circuit. Data are only presented for target [NO] of 20 ppm and frequency 10 Hz. Closed bars represent proximal sampling, grays bar represent middle sampling, and open bars represent distal sampling. A: pre-humidifier. B: post-humidifier. C: post-bellows.

Nitric oxide delivery systems have been thoroughly investigated with conventional mechanical ventilation by us and others.[7–10] However, the study of nitric oxide delivery during HFO is limited. Markhorst et al[14] investigated [NO] delivery during HFO and concluded that nitric oxide delivery with a standard rotameter and continuous titration into the standard circuit could result in an unpredictable [NO]. In their study, however, only mean delivered [NO] was evaluated and mixing of NO was not assessed because they used a chemiluminescence NO analyzer that had a relatively slow response time. Espinosa and Marks[15] evaluated NO delivery during HFO using a mass flow controller and reported acceptability of this method. However, they used a slow-response electrochemical analyzer that is unable to detect fluctuation of [NO]. It is important to use a fast-response NO analyzer when evaluating NO delivery systems because fluctuation of [NO] can be underestimated with a slow-response analyzer.[9] To our knowledge, ours is the first study investigating NO delivery during HFO with a fast-response NO analyzer. In addition, we adjusted NO flow without oscillation to avoid the effects of oscillation on the rotameter as reported by Markhorst et al.[14]

We found that the delivered [NO] can be lower than the target [NO] when NO is injected after the bellows of the oscillator with a hard circuit. This injection site was close to the pressure release valve. We speculate that some gas with higher [NO] than target was lost through this pressure release valve because of inadequate mixing and continuous leakage through this valve, which resulted in a lower delivered [NO]. Mixing of NO may be better with the soft circuit because [NO] was the same as target [NO], which means that gas lost through the pressure release valve was already well mixed. The soft circuit has a better shape for mixing of NO because of a larger mixing chamber and a circuit with a narrower caliber. It is also possible that we

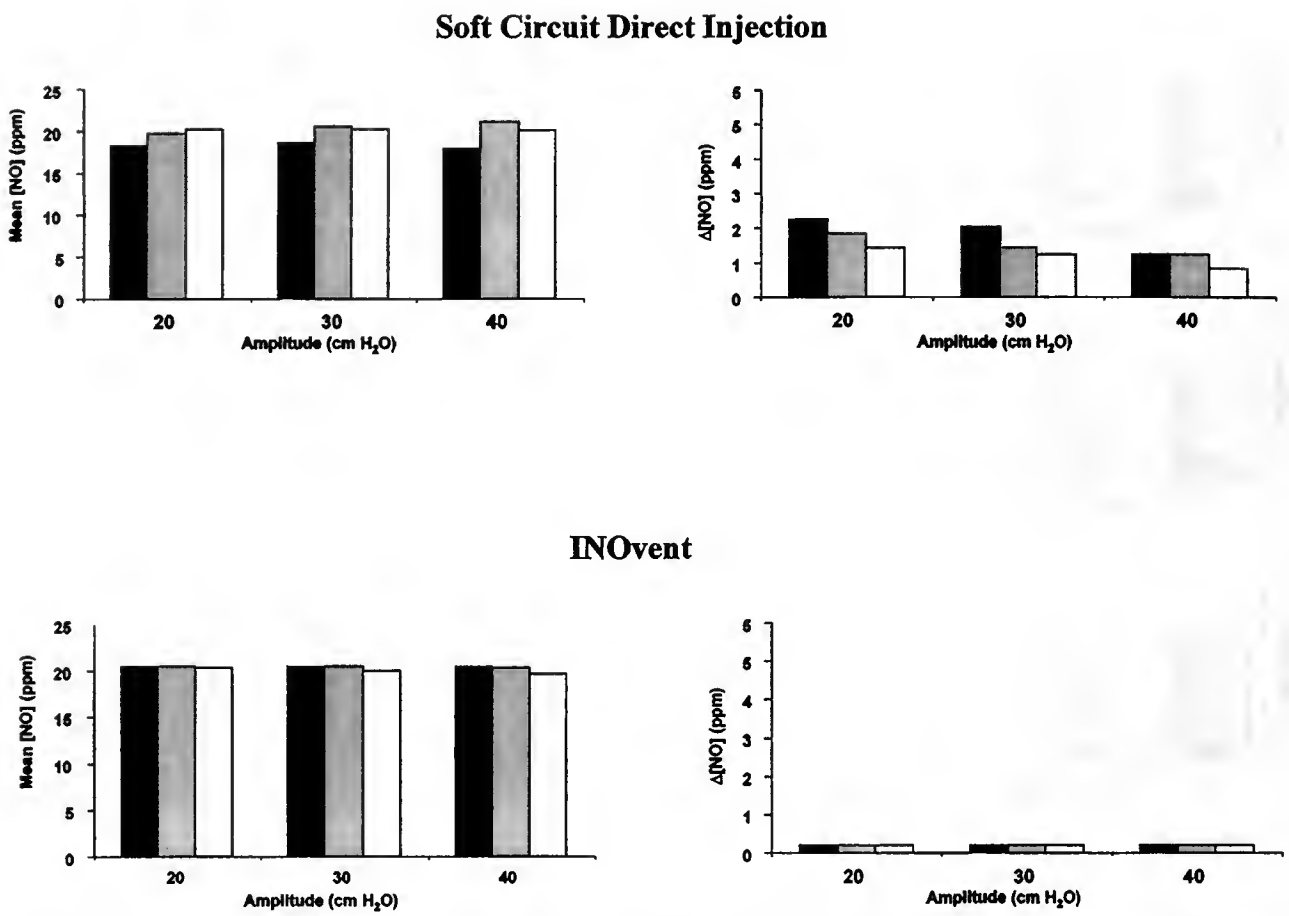

Fig. 5. Mean NO concentration ([NO]) and [NO] fluctuation (Δ[NO]) in parts per million (ppm) with mean airway pressure of 25 cm H_2O at each amplitude and at each sampling site with the soft circuit and with the hard circuit using the INOvent Delivery System. Only data from injection site C and frequency 10 Hz are presented for the soft circuit. Only injection site A was evaluated for the INOvent Delivery System. The closed bars represent proximal sampling, the gray bars represent middle sampling, and the open bars represent distal sampling.

Table 1. NO_2 Concentrations at Various HFO Settings

Set $\bar{P}_{AW}$ (cm H_2O)	Set P_{AMP} (cm H_2O)	[NO_2] (ppm)			$\bar{P}_{AW}$ (cm H_2O)	ΔP (cm H_2O)	$\dot{V}$ (L/s)	V_T (mL)
		A	B	C				
15	20	0.7	0.7	0.5	15.1	27.4	0.10	2.6
	30	0.5	0.5	0.4	15.2	41.9	0.14	3.2
	40	0.4	0.4	0.3	14.8	56.4	0.17	4.0
20	20	0.7	0.7	0.5	20.2	27.8	0.10	2.5
	30	0.6	0.5	0.4	20.0	42.6	0.14	3.2
	40	0.4	0.4	0.3	19.7	57.8	0.18	4.2
25	20	0.7	0.7	0.5	25.4	27.9	0.10	2.6
	30	0.6	0.6	0.4	25.2	43.6	0.14	3.2
	40	0.4	0.4	0.3	24.8	58.9	0.17	4.0

HFO = high frequency oscillation. $\bar{P}_{AW}$ = mean airway pressure. P_{AMP} = pressure amplitude. [NO_2] = concentration of nitrogen dioxide. ppm = parts per million. ΔP = pressure difference between highest and lowest airway pressure. $\dot{V}$ = inspiratory flow at the lung model. V_T = tidal volume delivery to the lung model. A: Pre-humidifier. B: Post-humidifier. C: Post-bellows. Data are presented only for 20 ppm NO.

sampled inadequately mixed gas with lower [NO] with the soft circuit because we observed a lower [NO] at the proximal sampling site.

Skimming et al[16] have shown that gas streaming can occur when NO is infused into a continuous flow of gas. Owing to this effect, the highest NO concentration is closest to the tubing wall nearest the infusion point, and the lowest concentration is between the tubing axis and the opposite tubing wall. Skimming et al[16] reported greater gas mixing with corrugated tubing than with smooth tubing, an effect that may be similar to the effect that we report with the soft circuit rather than with the hard circuit. Skimming et al[16] also found that gas mixing was virtually complete 30 cm from the point of NO infusion, a finding that is consistent with our results.

The measured [NO_2] was within the clinically acceptable range. The highest [NO_2] that we measured was 0.7 ppm, and there was a tendency for [NO_2] to decrease with a higher P_{AMP}. The test lung does not absorb delivered NO, which reacts with oxygen in the test lung. Therefore, it is possible that we overestimated NO_2 production during HFO in our study. We speculate that we observed a lower [NO_2] with higher P_{AMP} because of the enhanced washout of NO_2 in the presence of higher tidal volume (see Table 1). This is consistent with the previous report investigating NO_2 production during adult conventional mechanical ventilation.[17]

The INOvent is a commercially available NO delivery system that can deliver a reliable and stable [NO] during conventional mechanical ventilation.[12,13] In this study we found that the INOvent also delivers a reliable [NO] during HFO. Fluctuation of [NO] with the INOvent was less than with the standard rotameter with injection pre-humidifier. Although the difference was small, this may mean that flow delivery of NO by the INOvent is more stable than with the rotameter. We also found the INOvent Delivery System easy to use with HFO.

Imanaka et al[9] reported [NO] fluctuations of nearly 10-fold with continuous titration of NO into the ventilator circuit with an adult ventilator. This is because flow through the inspiratory circuit occurs during the inspiratory phase only, allowing NO to accumulate in the circuit during the expiratory phase. With the continuous flow that occurs during HFO, we found that the error with continuous titration of NO into the circuit was $< 10\%$. It is also important to point out that the fluctuation in [NO] was least when measured at the lung model. The clinical implication of this finding is that [NO] should be measured near the patient in the inspiratory limb of the circuit.

Although we found that NO can be delivered reliably during HFO, our results should not be extrapolated to high-frequency jet ventilation. The jet ventilator is used in tandem with a conventional ventilator. When conditions are such that the conventional ventilator interrupts the jet ventilator, large and unreliable concentrations of NO are delivered.[18] Thus, NO should not be delivered by jet ventilation until reliable methods are designed to accomplish this. This is likely to be problematic because of the difficulty of measuring [NO] distal to the point of gas injection into the airway (ie, the trachea).

Conclusions

In conclusion, mixing of NO in the bias gas flow during HFO using a continuous titration method is adequate with all of the injection sites that we evaluated. Injection before the humidifier is preferable, however, because this produced the smallest fluctuation of [NO]. The INOvent Delivery System provided a stable [NO] with HFO.

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Evaluation of a Fiberoptic Blood Gas Monitor in Neonates with Congenital Heart Disease

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BACKGROUND: Blood gas analysis is extremely important in perioperative management of neonates with congenital heart disease, where ventilator manipulation of the pulmonary vascular resistance is crucial. Delays in blood gas analysis resulting from transport of samples to a central laboratory may compromise management of these patients. Furthermore, neonates with congenital heart defects may have lower arterial oxygen (P_{aO_2}) levels due to intracardiac right-to-left shunting. We evaluated the Sensicath System in neonatal patients following cardiac surgery by simultaneously measuring specimens on the central laboratory blood gas analyzer. METHODS: After patients returned from the operating room, the Sensicath System was connected to the arterial line. Blood was pulled across the sensor and re-infused to the patient after analysis. The accuracy and precision of the Sensicath System blood gas analysis results were assessed by comparison to simultaneous samples analyzed with a Corning 855 analyzer. The specimen-result turnaround time was recorded. 97 samples from 5 patients were compared. RESULTS: Blood gas analysis results from the Sensicath System showed acceptable accuracy and precision: partial pressure of oxygen (P_{O_2}), $r^2 = 0.89$, bias = −4.5 mm Hg, precision = 11.8; partial pressure of carbon dioxide (P_{CO_2}), $r^2 = 0.59$, bias = −0.4 mm Hg, precision 6.2; pH, $r^2 = 0.78$, bias = 0.03 mm Hg, precision 0.03. The central lab specimen-result turnaround time was 13.8 ± 7.1 minutes. The Sensicath System provided results after a 60-second analysis time with no blood loss. CONCLUSIONS: When compared to a Corning 855 blood gas analyzer, the Sensicath System was found to provide acceptable blood gas values, with no iatrogenic blood loss. This system may be especially helpful in infants with congenital heart defects, since rapid results are necessary for optimal patient care. *Key words: fiberoptic, blood gas analyzer, monitor, neonate, congenital heart disease, iatrogenic blood loss, turnaround time.* [Respir Care 2000;45(9):1105–1112]

Background

Arterial blood gas (ABG) analysis, which provides detailed information on oxygenation, gas exchange, and acid-base balance, is one of the most frequent laboratory tests in the critical care environment.[1,2] Accuracy of ABG data are particularly important in the management of neonates with congenital heart defects, where ventilator manipulation of the pulmonary vascular resistance is crucial and because partial pressure of oxygen (P_{O_2}) values in this population often breach the lower limit of normal.[3] In neonates with congenital heart defects resulting in intracardiac shunting, ventilator management is often used to manipulate the pulmonary vascular resistance to optimally balance pulmonary and systemic blood flows.[4,5] Rapid blood gas analysis is integral to perioperative management of infants with heart disease to accurately assess gas exchange and oxygen delivery after ventilator changes.[6–9] Furthermore, this may result in frequent blood gas analysis, resulting in excessive iatrogenic blood loss.

In most centers, blood is drawn from a patient and transported to a central laboratory for processing. This may result in substantial time delays that can compromise patient management.[10,11] Over the last two decades, fiberoptic blood gas sensors have been developed that offer small electrode size and improved stability. This type of sensor

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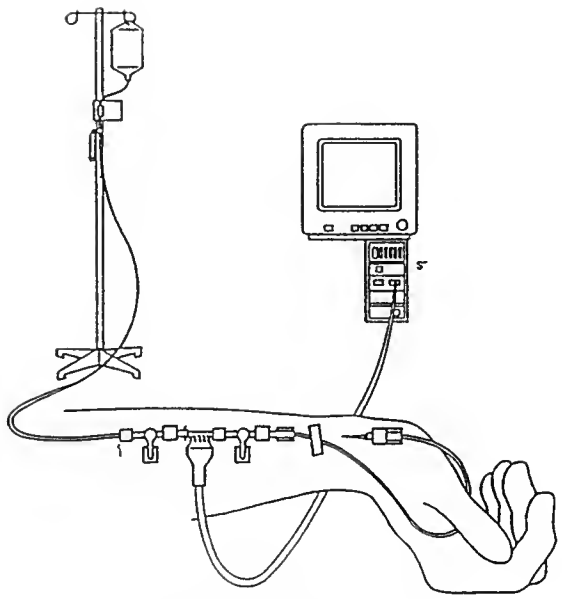

SensiCath Optical Sensor

Fig. 1. Sensor attachment to the arterial line. Proximal stopcock is closest to patient (laboratory specimen site), and distal stopcock is farthest from patient. (Courtesy of Optical Sensors Inc, Minneapolis, Minnesota.)

technology has been used in conjunction with continuous in vivo and on demand ex vivo monitoring. These blood gas systems permit rapid bedside monitoring of blood gases without permanent blood loss to the patient. In the present study, the accuracy and precision of the Sensicath System fiberoptic blood gas monitoring system (Optical Sensors Incorporated, Minneapolis, Minnesota) was assessed in postoperative neonates after cardiac surgery. The Sensicath System provides ex-vivo ABG results quickly, with no iatrogenic blood loss.

Device Description

The Sensicath System consists of a fiberoptic sensor, a blood gas module, and an OpticalCAM monitor. The fiberoptic sensor, which contains analyte-specific fluorescent dyes, is connected by fiberoptic cable to the OpticalCAM monitor. It is connected into the arterial line by standard Luer-Lok stopcocks. During analysis, blood is withdrawn from the arterial line past the sensor, and into the distal stopcock (see Fig. 1). The pH, arterial partial pressure of carbon dioxide (P_{aCO_2}), and arterial partial pressure of oxygen (P_{aO_2}) are measured spectrophotometrically and displayed on the monitor. After analysis, the blood is returned to the patient, and then the arterial line is flushed with heparinized normal saline.

Sensor calibration is performed prior to patient use and then daily. Calibration solutions are sterile, aqueous, nontoxic, and blood compatible. The Sensicath System ranges of measurement are 7.15–7.65 for pH, 20–80 mm Hg for P_{aCO_2}, and 40–600 mm Hg for P_{aO_2}.

Evaluation Methods

Neonates with congenital heart defects requiring corrective or palliative surgery were included in the study, as these neonates typically require frequent ABG analysis. Informed consent was obtained from the parents of all the infants prior to enrollment. The study was approved by the Institutional Review Board at Children's Hospital Medical Center.

The Sensicath System was connected to the patient's arterial line postoperatively. When blood gas analysis was required, blood was pulled across the fiberoptic sensor to allow measurement of pH, partial pressure of carbon dioxide (P_{CO_2}), and P_{O_2}. Blood was re-infused to the patient after results were obtained. During the Sensicath System analysis, a simultaneous arterial sample was drawn from the proximal stopcock and sent to the central laboratory for analysis. Laboratory samples were placed on ice, sent via pneumatic tube system to the central laboratory, and analyzed with a Corning 855 blood gas/electrolyte analyzer (Chiron Diagnostics, Norwood, Massachusetts). The specimen result turnaround time (defined as time from specimen draw to receipt of blood gas result in the cardiac intensive care unit) was recorded for each sample.

Ninety-seven samples analyzed simultaneously by the Sensicath System and Corning analyzer were compared. Bias and precision of the Sensicath System were determined by comparison with the Corning results using Bland-Altman analysis. Bias was defined as the mean difference between Sensicath System and Corning blood gas values. Precision was defined as the standard deviation of the mean difference between sensor and Corning blood gas measurements. In addition, bias and precision were assessed specifically for P_{O_2} values < 50 mm Hg. To supplement the Bland-Altman analyses, correlation between the data sets was also determined by linear regression analysis.

Results

Five patients (2 male, 3 female), each < 1 month old, were entered into the study. Cardiac diagnoses were: interrupted aortic arch (1), hemitruncus (1), hypoplastic left heart syndrome (2), and heterotaxy (1). Table 1 summarizes the diagnoses, number of blood gases, and ages at surgery.

Ninety-seven simultaneous blood gases measurements were obtained over a maximum period of 7 postoperative days. The ranges of values obtained were 7.31–7.57 for pH, 19–57 mm Hg for P_{CO_2}, and 27–134 mm Hg for P_{O_2}. Table 2 summarizes the results from individual patients.

Table 1. Patient Characteristics

Patient No.	Age at Surgery	No. Gases	Diagnosis	Surgery	Weight	Gestational Age
1	5 days	19	VSD, IAA	VSD closure	3.5 kg	39 weeks
2	3 days	21	DILV, AA, VSD	Norwood	3.6 kg	40 weeks
3	1 day	31	Heterotaxy, TAPVR, VSD, RV hypoplasia	BT shunt	3.7 kg	40 weeks
4	4 days	21	HLHS	Norwood	1.8 kg	34 weeks
5	18 days	5	Hemitruncus	Reimplant	3.6 kg	40 weeks

VSD = ventricular septal defect. IAA = interrupted aortic arch. DILV = double-inlet left ventricle. AA = aortic atresia. TAPVR = total anomalous pulmonary venous return. RV = right ventricle. BT shunt = Blalock-Taussig shunt. HLHS = hypoplastic left heart syndrome.

Table 2. Individual Patient Data

Patient No.	Measured By/Bias/Precision	pH	P_{CO_2} (mm Hg)	P_{O_2} (mm Hg)	P_{O_2} < 50 mm Hg
1	Lab	7.41	40	107	NA
	Sensor	7.45	39	92	NA
	Bias	0.06	−1.4	−13.56	—
	Precision	0.11	2.08	17.72	—
2	Lab	7.38	48	44	41
	Sensor	7.39	48	43	47
	Bias	0.01	−0.41	1.96	5.23
	Precision	0.03	−0.49	3.56	10.45
3	Lab	7.41	45	44	43
	Sensor	7.46	46	42	42
	Bias	0.07	0.91	−1.06	−2.03
	Precision	0.08	5.73	3.31	4.65
4	Lab	7.5	30	101	NA
	Sensor	7.5	25	90	NA
	Bias	−0.03	−5.3	−11.3	—
	Precision	0.03	4.8	11.3	—
5	Lab	7.4	42	40	39
	Sensor	7.43	42	33	33
	Bias	0.04	−0.58	−5.79	−6.56
	Precision	0.23	4.22	5.82	6.68

P_{CO_2} = partial pressure of carbon dioxide. P_{O_2} = partial pressure of oxygen. NA = not applicable.

Table 3 shows the bias and precision for each variable. Bias and precision for the pH sensor were excellent (0.03, 0.03), indicating trivial bias between the two systems. Sensicath System P_{CO_2} performance also showed minimal bias (−0.40). The P_{O_2} measurement bias was slightly greater (−4.5), but still clinically unimportant. The precisions of the P_{CO_2} and P_{O_2} sensor measurements were 6.2 and 11.8, respectively. The bias and precision of P_{O_2} values < 50 mm Hg were slightly better (−1.6 and 5.6). Figure 2 shows Bland-Altman plots for all values. Figure 3 shows linear

Table 3. Sensor Results

Variable	*n*	Bias	Precision	r^2	p value
P_{O_2} (mm Hg)	97	−4.5	11.8	0.89	< 0.0001
P_{O_2} < 50 mm Hg	65	−1.6	5.6	0.25	NS
P_{O_2} (mm Hg)	97	−0.40	6.2	0.59	NS
pH	97	0.03	0.03	0.78	< 0.05

P_{CO_2} = partial pressure of carbon dioxide. P_{O_2} = partial pressure of oxygen. NS = not significant.

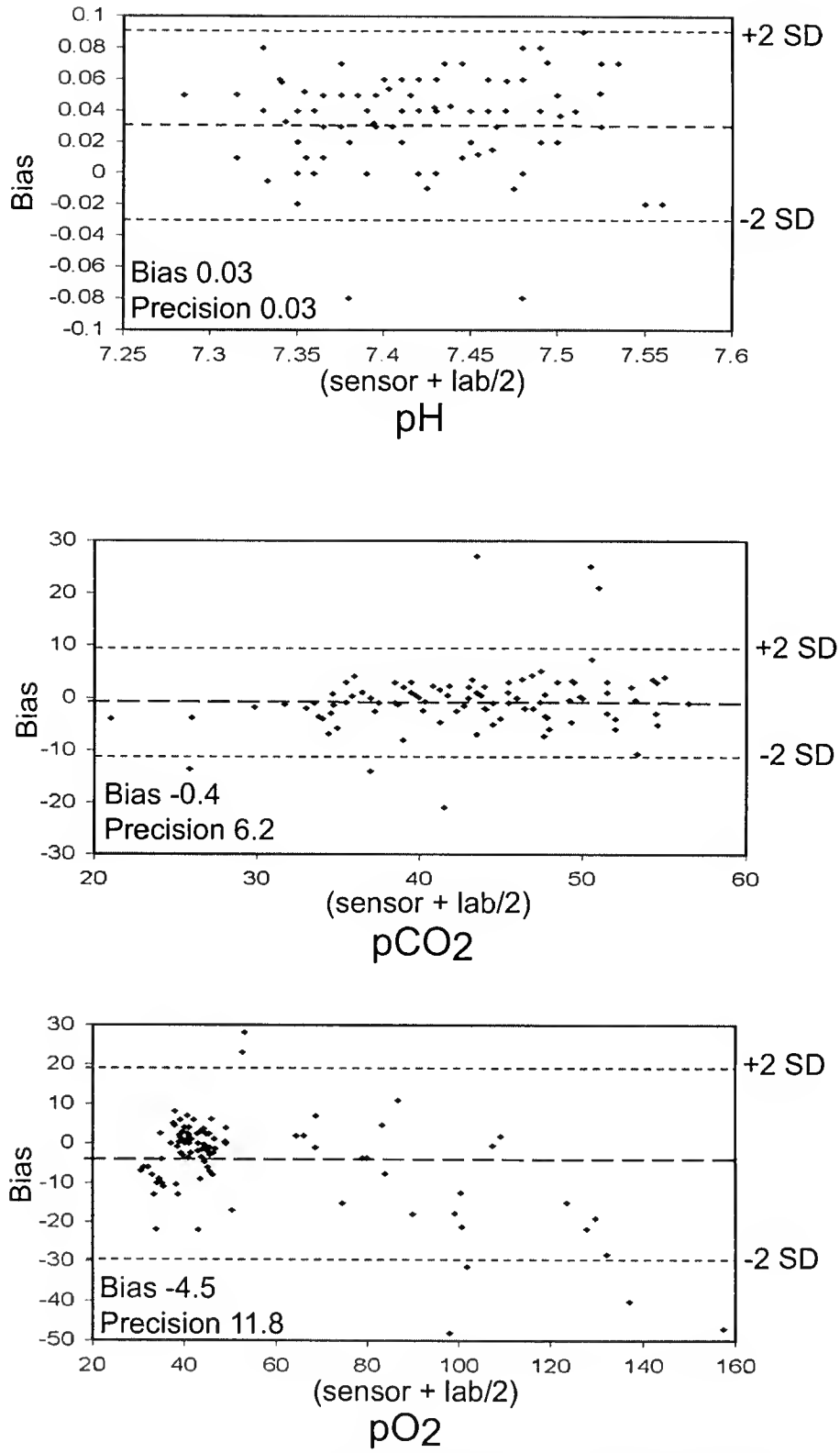

Fig. 2. Bland-Altman plots of the bias and precision for pH, partial pressure of carbon dioxide (P_{CO_2}), and partial pressure of oxygen (P_{O_2}) for all patients. Bias represents the mean difference for all samples. The majority of values fall within the limits of agreement (±2 standard deviations), as indicated by the outer (dashed) lines.

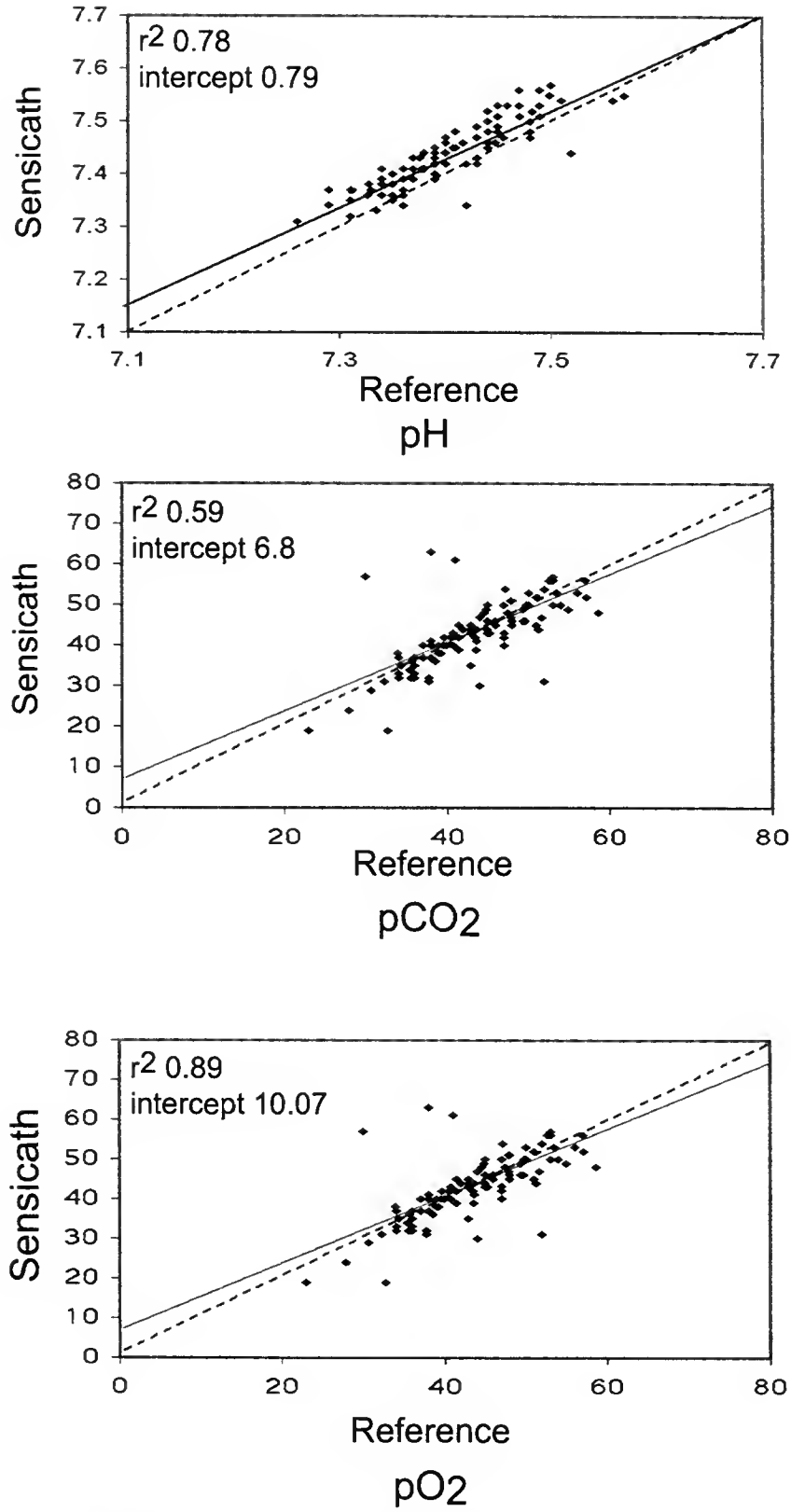

Fig. 3. Linear regression for all values, using Corning 855 blood gas and electrolyte analyzer as the reference. P_{CO_2} = partial pressure of carbon dioxide. P_{O_2} = partial pressure of oxygen.

Table 4. Duration of Device Utilization

Variable	Bias/Precision	First 24 h	24–48 h	48–72 h	> 72 h
pH	Bias	0.03	0.03	0.04	0.04
	Precision	0.03	0.03	0.02	0.03
P_{CO_2}	Bias	−1.06	2.16	1.05	−2.15
	Precision	5.21	7.90	8.44	4.06
P_{O_2}	Bias	−7.42	−0.4	−1.44	−5.16
	Precision	11.39	11.4	6.36	13.14
$P_{O_2} < 50$ mm Hg	Bias	−3.71	−2.16	−0.05	−0.04
	Precision	5.65	7.78	2.81	4.31

P_{CO_2} = partial pressure of carbon dioxide. P_{O_2} = partial pressure of oxygen.

regression analyses for each variable to show direct correlation of the data sets.

The central laboratory provided results with a turnaround time of 13.8 ± 7.1 minutes. The Sensicath System provided results after a 60-second analysis time, without patient blood loss.

Device use ranged from 26 hours to 132 hours. The average length of sensor use was 84.6 ± 43.4 hours. Table 4 summarizes bias and precision data based on the length of sensor use. As sensor use increased, sensor accuracy and precision improved. The number of ABGs obtained per length of sensor utilization was 6.4 ± 2.2 for the first 24 hours of use, 4.6 ± 2.9 for 24–48 hours of sensor use, 3.75 ± 1.3 for 48–72 hours of sensor use, and 9.0 ± 4.6 for sensors used > 72 hours.

Discussion

Standards for the accuracy and precision of blood gas monitors do not currently exist, so the results of this study were evaluated based on the Clinical Laboratory Improvement Act (CLIA) 1988 standards for bias and precision of blood gas analyzers, which are:

- pH: bias 0.05, precision ± 0.04
- P_{CO_2}: bias 3.0 mm Hg, precision ± 5.0 mm Hg
- P_{O_2}: bias 5.0 mm Hg, precision ± 8.0[12]

The data indicate excellent agreement between the Sensicath System and laboratory pH measurements, meeting recommended standards. The bias of the P_{CO_2} sensor was also excellent, although the precision was slightly above CLIA 1988 recommendations. The Sensicath System manufacturer's specification for oxygen accuracy is ±10% between 51 and 280 mm Hg. In this study, the Sensicath System was tested within and below the manufacturer's accuracy specifications. The P_{O_2} values showed low levels of bias, meeting CLIA 1988 standards. Precision was marginally acceptable, however, falling outside recommended guidelines. The observed variability occurred in only a small number of paired samples, however, and the analysis did not determine whether the variability resulted from the Sensicath System measurement, blood gas analyzer measurement, or sample transport to the central laboratory (Figs. 2 and 3). Furthermore, a limitation of the present study was the small number of patients evaluated. Evaluation of more patients may have yielded better bias and precision values.

Measurement of P_{O_2} values < 50 mm Hg is particularly relevant when caring for patients with congenital heart disease. Neonates with congenital heart defects often have low P_{O_2} values because of intracardiac right-to-left shunting of blood.[11] In 3 of the 5 patients in this study, the majority of P_{O_2} values (n = 65) were < 50 mm Hg. The Sensicath System performed well in this low range; the bias and precision for this group of data were within recommended standards (Fig. 4). Although correlation for this group of patient results was fairly low (0.25, see Fig. 4b), this was due to the relatively narrow range of P_{O_2} values in this subset of samples. Small differences of 1–5 mm Hg have a significant effect on overall correlation, but are of limited clinical importance.

Other fiberoptic blood gas monitors have been evaluated clinically. The results of our study compared favorably with results obtained by Zimmerman and Dellinger[13] (pH: bias −0.021, precision ± 0.037; P_{CO_2}: bias 1.74 mm Hg, precision ± 6.06; P_{O_2}: bias −5.89 mm Hg, precision ± 0.037), McKinley and Parmley[14] (pH: bias 0.13, precision ± 0.031; P_{CO_2}: bias 1 mm Hg, precision ± 3.1; P_{O_2}: bias 5.7 mm Hg, precision ± 7.7), and Venkatesh et al[15] (pH: bias 0.075, precision ± 0.450; P_{CO_2}: bias 1.65 mm Hg, precision ± 4.95; P_{O_2}: bias 4.5 mm Hg, precision ± 20.78). Zimmerman and Dellinger[13] performed blood gas measurements on a bedside monitor and two blood gas analyzers simultaneously (same manufacturer/model) using split sample testing. The bias and precision between the two blood gas analyzers was similar to that observed between the blood gas monitor and blood gas analyzers. Other studies have also demonstrated that significant vari-

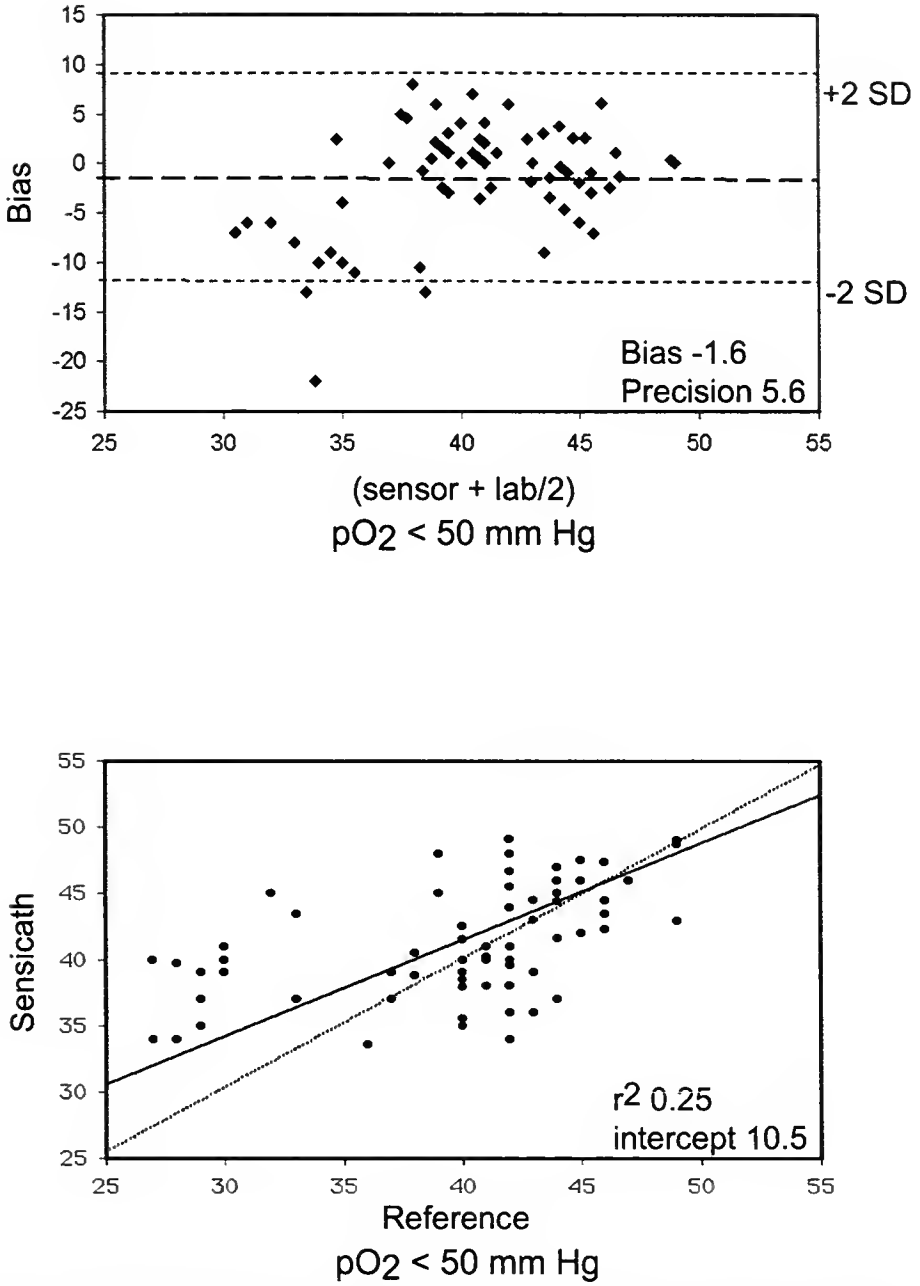

Fig. 4. Bias and precision plots reveal that most results fall within 2 standard deviations of the mean. Reference is the Corning 855 blood gas and electrolyte analyzer. P_{O_2} = partial pressure of oxygen.

ability can exist among measurements made by different blood gas analyzers.[16,17]

Blood gas analysis result variability may be explained by several factors. Red blood cell sedimentation, specimen dilution, air bubbles, excessive heparin in a sample, or a combination of these factors can cause measurement inconsistency. Protein build-up within the sensor can lead to measurement errors, but can be avoided by flushing the sensor regularly with heparinized saline. Since specimens sent to the central laboratory may have greater risk of inaccurate results, analysis by a bedside blood gas monitoring system may improve accuracy.[18]

The greatest benefit of bedside blood gas monitoring is rapid access to specimen results. The Sensicath System provided accurate blood gas results within 60 seconds. This is particularly valuable in infants with complex congenital heart disease where ventilator manipulation of pulmonary vascular resistance is crucial to optimize systemic

oxygen delivery. Transport to a central laboratory for analysis can result in excessively long turnaround times, thus compromising patient care.[19] Recent surveys indicate that physicians desire a turnaround time of ≤ 10 minutes on all ABGs.[20] The American Heart Association and the Emergency Care Research Institute have recommended turnaround time standards of ≤ 5 minutes during open heart surgery and critical patient care situations.[21] A delay may impede patient care or necessitate repeat analysis if the delayed results are no longer clinically relevant. Unfortunately, many central laboratories are unable to provide this type of service.[21] Specimen results turnaround time is defined as the time from blood draw to the time results are available. There can be discrepancies between hospital laboratories and intensive care units in reporting turnaround times.[22] Many laboratories report turnaround time as the time the specimen is received to the time the results are entered into the hospital's laboratory information system.[23] Since specimen transport can increase the actual turnaround time by as much as a third, the turnaround time in some laboratories can be much longer than reported.[24]

The other major benefit of the Sensicath System is elimination of iatrogenic blood loss. Blood loss in conventional blood gas analysis results from sampling and from clearing indwelling lines prior to sampling. Thirty percent of ICU phlebotomy blood loss results from the discard of blood and heparin mixtures in order to clear indwelling lines.[25] The Sensicath System eliminates iatrogenic blood loss, which may reduce the need for blood transfusions.[26]

Conclusions

The Sensicath System was found to provide accurate blood gas results rapidly, with no iatrogenic blood loss to the patient. This system may be especially helpful in patients with complex heart defects, where ventilator manipulation of pulmonary vascular resistance is used to optimally balance pulmonary and systemic blood flows.

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Left Hemithorax Opacification in a Term Newborn Infant

Katherine A Douglas RRT and Brian S Bradley MD

Case Summary

A term male infant was born to a 26-year-old woman following an uncomplicated pregnancy. All prenatal lab studies, including those for sexually transmitted diseases, were negative. There was no report of fetal sonography. The mother works in the distribution center of a department store; the father is a custodian at a local university. Both parents are smokers: the mother smokes between one half and one pack per day. There was a direct but distant family history of known cancer. The paternal great-grandparents had unspecified cancer; breast and skin cancer were reported in a maternal great-grandmother.

The 3.49 kg infant was born at an outlying hospital by vaginal vertex delivery complicated by a difficult vacuum extraction. The baby presented blue, floppy, and with poor ventilatory effort. The baby's own respiratory efforts were marked by expiratory grunting and lack of air movement auscultated in the chest. The infant's color failed to improve in spite of positive pressure ventilation with 100% oxygen via bag and mask. The Apgar score was 4 at one minute of age and 5 at five minutes of age. Figure 1 shows the initial chest radiograph.

Fig. 1. Anteroposterior chest radiograph of the patient shortly after birth.

Questions

What is the general appearance of the chest in Figure 1?

What is the position of the trachea?

What should be included in the differential diagnosis of this patient?

What further diagnostic imaging is indicated?

Answers

There is no rotation of the patient on the anteroposterior chest radiograph. There is dense opacification of the entire left hemithorax. There are minimal lung markings and overall reduced lung aeration in the right hemithorax with deviation of both the trachea and the esophagus to the right.

The differential diagnosis of respiratory distress in the newborn associated with marked one-sided density on radiograph includes a chest mass (eg, neuroblastoma, teratoma), congenital cystic adenomatoid malformation, pulmonary sequestration, congenital diaphragmatic hernia, or a pleural effusion.

A computed tomography (CT) scan is indicated for further diagnosis.

Discussion

Resuscitation and stabilization of newborns presenting with central cyanosis and poor tone at delivery dictates a series of initial steps well addressed in the Neonatal Re-

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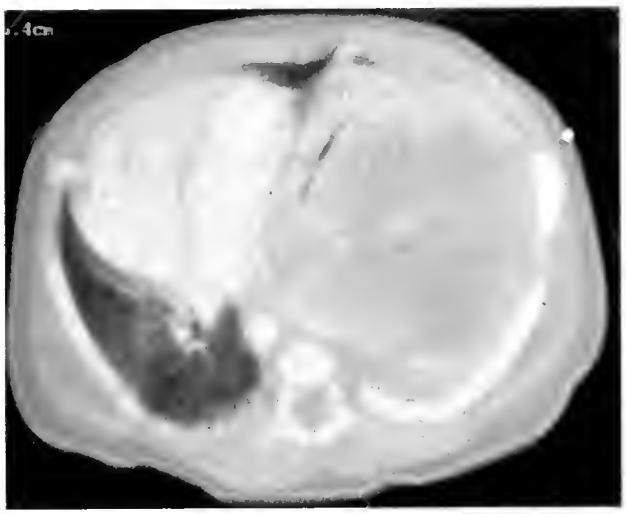

Fig. 2. Computed tomography image of the chest at mid-thorax, showing the heart displaced to the right and the partially inflated right lung. The left hemithorax is filled with a heterogeneous mass and no aerated lung is visible.

suscitation Program.[1] After the initial chest radiograph was taken, this infant was successfully resuscitated, but a CT scan (Fig. 2) was immediately scheduled following transport to our referral center.

Pulmonary neoplasms in the newborn are rare.[2] When an opacity presents on a neonate's chest radiograph, the more common abnormalities should be suspected. These include intrapulmonary lesions such as cystic adenomatoid malformation of the lung, which is a congenital cystic lung anomaly. Extrapulmonary lesions such as chest wall mass or pleural effusion (usually associated with hydrops fetalis) should also be included. Pulmonary sequestration (lack of connection between the lung tissue and bronchial tree and/or vascularity) should be included in the early differential diagnosis as well.

Both CT and magnetic resonance imaging scans have demonstrated usefulness in the diagnosis of pediatric chest masses. The CT scan renders information pertinent to the morphology, localization, and the extension of masses or tumors.[3] The CT scan done on our patient (see Fig. 2) showed a solid nonhomogenous mass with some irregular areas of fluid density within it. Midline structures were displaced and the heart was shifted to the right. No lung could be seen on the left; posteriorly a small amount of pleural fluid was seen.

When the patient was taken to the operating room, the mass was found to be solid and noncompressible. The tumor was excised in its entirety. The microscopic and immunohistochemical findings on pathology examination were consistent with congenital infantile fibrosarcoma.

Congenital infantile fibrosarcoma is a rare malignant tumor occurring in newborn infants, representing less than 1% of childhood cancers.[4] The tumor is usually a soft tissue tumor of the extremities,[5,6] but there have been isolated cases recently reported of this tumor presenting in the chests of neonates.[2,7,8] Excision is the appropriate treatment for these fibrous tumors.[4] The use of chemotherapy and/or radiotherapy following surgery is controversial, since many of these tumors are not malignant.[4,9,10] The prognosis for infants born with this type of tumor is considered to be very good.[2,4,7]

Our patient had a benign recovery following a complete, left-sided pneumonectomy. He was discharged to home on day 20 of life. He is being followed by our pediatric hematology/oncology service for subsequent chemotherapy.

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Occult Carboxyhemoglobinemia and Hypoxemia in a Patient with Malaria

Jeffrey M Haynes RRT RPFT and James T St Pierre RRT RN

Determination of arterial oxygenation is an important form of pulmonary function testing, whether the measurements are made in or out of the pulmonary function laboratory. The choice between obtaining an arterial blood gas (ABG) measurement or functional oxygen saturation level via pulse oximeter (S_{pO_2}) to assess oxygenation not only has cost and morbidity implications; clinical conclusions and treatment decisions can differ greatly if an arterial oxygen saturation disparity exists (ie, oxyhemoglobin [O_2Hb] and S_{pO_2} differ substantially). The following case illustrates how an O_2Hb-S_{pO_2} disparity can affect the decision of whether to administer oxygen therapy.

Case Summary

A 17-year-old nonsmoking male patient presented to the hospital with generalized weakness, vomiting, diarrhea, fever, and rigors 2 days after returning from a 3-week stay in Tanzania. Because of his religious beliefs, malaria chemoprophylaxis had not been administered. A malaria infection was suspected, and a diagnosis of malaria *falciparum* infection was established following microscopic examination of the patient's blood. By hospital day 4 the patient's protozoal infection had resulted in renal insufficiency, metabolic acidosis, hemolytic anemia, and thrombocytopenia. An ABG was ordered to assess acid-base status; at the time of ABG sampling the patient was not being treated with supplemental oxygen because his S_{pO_2} was 95%. Table 1 lists the results of the ABG analysis.

Table 1. Results of Arterial Blood Gas Analysis in a Malaria Patient

Measurement	Value
pH	7.42
P_aCO_2 (mm Hg)	24
P_aO_2 (mm Hg)	59
HCO_3 (mmol/L)	15.4
BE (mmol/L)	−8.2
Hb (g/dL)	5.6
O_2 Hb (%)	89
COHb (%)	4.5
MetHb (%)	1.8
RHb (%)	4.7
Ca_{O_2} (mL/dL)	7.1

P_aCO_2 = arterial partial pressure of carbon dioxide
P_aO_2 = arterial partial pressure of oxygen
H_{CO_3} = bicarbonate
BE = base excess
Hb = hemoglobin
O_2 Hb = oxyhemoglobin
COHb = carboxyhemoglobin
MetHb = methemoglobin
RHb = reduced hemoglobin
Ca_{O_2} = oxygen content in arterial blood

Questions

What is the correct interpretation of the blood gas data listed in Table 1?

What is the primary cause of the O_2Hb-S_{pO_2} disparity?

What is the source of the patient's carboxyhemoglobinemia?

Answers

The ABG data reveal hypoxemia and mild carboxyhemoglobinemia; the source of the carboxyhemoglobin (COHb) is endogenous secondary to hemolysis. An O_2Hb-S_{pO_2} disparity developed due to the increase in COHb.

Discussion

Carbon monoxide (CO) is a notorious gas molecule widely regarded as an environmental pollutant and deadly poison generated from incomplete combustion of carbon. Yet this so-called "silent killer" is also produced endogenously by mammals, and, not unlike a more renowned environmentally and endogenously produced gas mole-

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cule, nitric oxide, CO plays an important role in the maintenance of homeostasis. Some of CO's physiologic functions include serving as a modulator of vascular tone,[1] uterine contractility during pregnancy,[2] carotid body sensory activity,[3] and as a neuronal messenger.[4] The endogenous production of CO occurs when heme released from destroyed erythrocytes is converted to biliverdin and CO by heme oxygenase.[5] The endogenous production of CO is in part responsible for the normal, small amount of COHb found in humans, which is usually < 2%. Hemolytic anemia is characterized by accelerated erythrocyte destruction, which increases CO production and COHb. The degree of hemolysis-related carboxyhemoglobinemia encountered in this case is consistent with other reports in the literature. Slusher et al[6] studied 55 jaundiced Nigerian infants and found COHb levels ranging from 0.43% to 5.93%, with higher COHb levels associated with greater need for exchange transfusion and higher incidence of kernicterus and death. Entman et al[7] used COHb as a surrogate for hemolysis in patients with preeclampsia and found COHb levels ranging from 1.6% to 4.1%. Though an endogenously-derived increase in COHb is not likely to cause symptoms such as those associated with severe smoke inhalation, the small increase in COHb observed in this case was enough to create spurious S_{pO_2} readings that proved to be clinically important.

Commonly used pulse oximeters use two wavelengths of light (660 nm and 940 nm) to determine the ratio of O_2Hb to reduced hemoglobin. At 660 nm, light absorption by O_2Hb and COHb are similar; therefore, increasing carboxyhemoglobinemia may preserve acceptable S_{pO_2} values in the presence of falling O_2Hb.[8,9] Since light absorption by O_2Hb and COHb at 940 nm are not similar, the extent that S_{pO_2} overestimates O_2Hb in the presence of COHb should not be expected to equal the concentration of COHb.[9] Though the effect of COHb on S_{pO_2} is fairly well known, it is probably not as well appreciated that the source of CO can be endogenous. Craft et al[10] reported the effects of endogenous CO on S_{pO_2} in children during sickle cell crises. In their study, in which S_{pO_2} and O_2Hb were measured simultaneously, S_{pO_2} was found to overestimate O_2Hb by an average of 6.9%, which was related to the COHb concentration. In that cohort of children with acute sickle cell disease, COHb ranged between 3.2% and 7.6%. In addition to COHb, the severe anemia present in our patient can also affect pulse oximetry accuracy; however, anemia tends to erroneously lower S_{pO_2} values.[11]

In summary, this report illustrates how reliance on pulse oximetry to assess oxygenation in a patient with unappreciated carboxyhemoglobinemia due to hemolytic anemia can lead to an ill-advised decision to withhold supplemental oxygen from a hypoxemic patient with critical illness. Clinicians should consider the potential influence of endogenously-derived COHb on S_{pO_2} when caring for patients with hemolytic disorders.

Acknowledgement

The authors would like to extend gratitude to Cindy Sloan, medical librarian, for her assistance

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Letters

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International Units for Hemodynamic Monitoring

It was very interesting for a European mechanical engineer, fully familiar with the Système international d' unités (the International System of Units, abbreviated SI) , to read Robert R Fluck Jr's letter (Respir Care 1998: 43:656).

Of course, it is obvious that he is right, and I see no reason why any scientist in the world would not admit his point immediately and take actions to correct this error.

I would like to make another comment:

A. In international units, ΔP is expressed in Pa (pascals). The use of bar or mbar is tolerated. But the use of dyn/cm^2 was abandoned a long time ago.

B. A flow $\dot{Q}$ is expressed in $m^3 \cdot s^{-1}$. The use of $L \cdot s^{-1}$ is tolerated. The use of $cm^3 \cdot s^{-1}$ is possible for smaller flows.

C. The units for a flow resistance should therefore be expressed in $Pa \cdot m^{-3} \cdot s$, in which the presence of Pa reminds that a pressure is involved in the process. It is, to my point, better than to break up the pressure Pa into $N \cdot m^{-2}$, wherein the unit of force doesn't reflect the nature of the phenomenon.

- The unit so defined is too small. For practical reasons, I prefer $Pa \cdot cm^{-3} \cdot s$.
- Converting $dyn \cdot s \cdot cm^{-5}$ into $Pa \cdot cm^{-3} \cdot s$ is simple:

$$1 \text{ dyn} \cdot s \cdot cm^{-5} = 10^{-1} \text{ Pa} \cdot cm^{-3} \cdot s$$

- The presence of a factor 10^{-1} will change the habits of the practitioners and might lead to errors. Let us therefore consider something else.

D. A pressure can be expressed in mbar (tolerated by the International Organization for Standardization [ISO]). A volume can be expressed in L (tolerated by ISO).

I would therefore propose to use $mbar \cdot L^{-1} \cdot s$. The conversion is now straightforward:

$$1 \text{ dyn} \cdot s \cdot cm^{-5} = 1 \text{ mbar} \cdot L^{-1} \cdot s$$

The advantages being that:

1. The units are internationally recognized and comply with the recommendations of the ISO.

2. The unit can be broken up into two parts: mbar for ΔP and L/s for $\dot{Q}$, which are both very common units and reflect the nature of the quantity measured.

From a pedagogical point of view, it is very interesting.

3. The dimension of this unit is the same as the previous one. No conversion factor. No error possible.

4. There are no high powers such as (-5) in the unit, which tend to frighten the students and to make this notion appear to be very difficult.

I hope this modest contribution will help.

Michel Bardel
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The author responds:

I very much appreciate Mr Bardel's comments. I am in favor of anything that will (1) simplify these confusing and intimidating units for resistance and (2) help people better understand the concept they are discussing. The fact that $mbar \cdot L^{-1} \cdot s$ is the same unit (conversion factor of 1) makes it very nice. The only problem continues to be the SI-phobic nature of the people in the United States. We need to move forward with "metrification" and join the rest of the world; people are resistant because it is change and because they are not familiar with the units. I believe I'll begin using these units when I teach our students about pulmonary and systemic vascular resistance in cardiovascular physiology class this fall.

Thanks, Mr Bardel, for a thought-provoking and useful letter.

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Reviews of Books and Other Media. Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104.

Books, Films, Tapes, & Software

The Lung at Depth. Claes EG Lundgren and John N Miller, Editors. (Lung Biology in Health and Disease, Volume 132, Claude Lenfant, Executive Editor.) New York/ Basel: Marcel Dekker. 1999. Hardcover, illustrated, 686 pages, $225.

Interest in diving is at an all time high—whether for sport, occupational, military, or scientific reasons. Deep diving records are also assaulted on a regular basis; for instance, the breath-holding depth record has been broken no less than 25 times in the last 50 years. Currently, records stand at over 700 m (over 2,300 ft) and 133 m (over 440 ft) for compressed gas and breath-hold diving, respectively. These facts imply that not only are increasing numbers of people, of all ages and states of health, participating in diving, but many are wanting to do so at depths greater than ever before.

The depths to which humans now venture are truly astounding, and yet the physical environment at depth represents an extreme challenge to human physiology, especially respiratory function. Consider the fact that divers reaching record depths face changes in ambient barometric pressure of greater than 13-fold for breath-holding, and greater than 70-fold for compressed air breathing! At these ambient pressures, many normally innocuous and unimportant factors (such as gas density and composition) severely limit physical performance, or worse, become potentially lethal. Though nearly everyone is familiar with the "bends," there are also a number of less recognizable hazards associated with diving. A few examples include: pre-existing medical conditions such as asthma or difficult-to-diagnose congenital pulmonary defects, either of which predispose those affected to potentially fatal complications (barotrauma) when resurfacing; repetitive diving, which, over a lifetime, may dispose those involved to permanent, long-term lung function changes, even in the absence of obvious injury mechanisms; and, finally, extremely cold water temperatures, often encountered at depth, which place lives in jeopardy by cooling breathing gases or interfering with the proper operation of a diver's underwater breathing apparatus.

But, just how much do we really know about potential diving hazards, scientifically and medically speaking? More importantly, how accurately can we predict just who is at risk while diving, and when? Do the authorities all agree upon who is at risk? How deep is too deep? This book explores and attempts to answer these and many other related questions. Moreover, the book details what is known about the changes to pulmonary physiology and the adaptations that must occur while diving.

The goal of this volume is to provide the reader with an authoritative compilation of what is currently known about respiratory function during and following diving. To this end, it includes over 1,800 literature citations. It begins with a chapter, "Respiratory Function at Depth," that explores the alterations to pulmonary architecture and gas behavior and how these can interact to limit air flow while breathing compressed gases at depth. Be prepared: many of the explanations in this and other chapters require a considerable background in mathematics and mathematical modeling. In Chapter 2, "Pulmonary Fitness for Diving," existing data are used to examine the current thinking on, and controversies involved in deciding precisely who is and who is not "fit to dive." For those with asthma and dismayed by having been advised never to dive, data presented in this chapter may change your mind. The physiologic effects experienced during partial and total immersion in water followed by those precipitated by changes in water temperature are discussed in Chapter 3, "Immersion Effects," and Chapter 4, "Temperature Effects." What we know about the ability of the body to cope with gas exchange when compressed gases are respired at depth is explained in Chapter 5, "Oxygen Toxicity," and in Chapter 6, "Carbon Dioxide Retention." Other important hazards associated with lung function and diving are considered in Chapter 7, "Gas Bubbles and the Lungs," Chapter 8, "Pulmonary Barotrauma," Chapter 9, "Long-Term Pulmonary Effects of Diving," and Chapter 10, "Drowning and Near-Drowning." The remaining 4 chapters focus on other interesting aspects of diving. In Chapter 11, "Liquid Breathing and Breath-Holding," the author discusses the current state of the art, as well as the potential for eventually using liquids in association with underwater breathing apparatuses or breath-hold diving. In a remarkably complete discourse, Chapter 12, "Underwater Breathing Apparatus," discusses the theory, standards, safety requirements, and engineering challenges associated with underwater breathing apparatuses. In another compelling chapter, Chapter 13, "Human Breath-Hold Diving," the authors relate just how little we really understand about the incredible physiologic adaptations that must occur during human breath-hold diving. For example, breath-hold divers now easily exceed depths once believed sufficient to collapse human lungs to a volume well below the functional residual volume; for most people this was predicted to occur at a depth somewhere between 30 and 50 meters of seawater (100–165 ft). At these depths and ambient pressures, if intrapulmonary lung volume momentarily falls below functional residual volume, a relatively negative pressure instantly develops within the airways, which is in turn followed by a rapid filling of the alveoli and pulmonary interstitium with edema fluid. For a breath-hold diver, this scenario would probably mean loss of consciousness under water and certain death—yet few die; this book tries to explain the protective physiologic phenomena involved, while recognizing that scientists have not yet developed a comprehensive and universally satisfactory explanation. The book ends with Chapter 14, "Diving Animals," which reviews what is known about the physiology of diving and adaptations among vertebrate animal divers such as turtles, whales, penguins, seals, and dolphins.

The target audience for this monograph is clearly serious scientists and engineers (both might be interested in what has been studied and by whom), and possibly some physicians clinically responsible for hyperbaric medicine at their institutions. For this relatively small group, the book is potentially an invaluable and definitive reference. Unfortunately, I believe it offers only a limited appeal to the average respiratory ther-

apist. Not only is the price exorbitant, it contains little or no information that might prove useful to a therapist interested in improving his or her skills—even those involved with hyperbaric medicine. Furthermore, the book provides virtually no background or basic information; rather, it presumes the reader has at least a working knowledge of respiratory physiology, mathematics, physics, engineering, and hyperbaric medicine. There are a couple of possible exceptions: therapists extremely interested in diving or in performing some forms of hyperbaric research might find it useful; and the chapters on barotrauma and drowning and near-drowning provide valuable insights into those topics and currently employed therapeutic strategies.

Though the content of this book is generally superior, the materials used in its construction are not; the cover and paper are, at best, average—hardly in accord with the cover price. To its credit, the book contains a plethora of equations, graphs, tables, and photographs; most of these are relatively easy to follow and very helpful in conceptualizing the sometimes esoteric or complicated topics.

All in all, this a very good book; it is well written and exceedingly thorough. It is nearly free of typographical or other editing errors. Certainly the expertise of the contributors cannot be questioned. For these reasons, I enthusiastically recommend it, but only for those specifically involved with, or interested in specific topics covered within this volume; others are cautioned to seriously consider the content, cost, and construction materials before making plans to purchase.

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CPR: Resuscitation of the Arrested Heart. Max Harry Weil MD and Wanchun Tang MD, Editors. Philadelphia: WB Saunders. 1999. Hardcover, illustrated, 318 pages, $58.

Cardiac disease remains the leading cause of death among adults in the United States, and sudden cardiac arrest is its most common manifestation. This book is a comprehensive and up-to-date summary of cardiac arrest and its management.

It is pointed out by the authors in the preface that there has been relatively little improvement in survival from cardiac arrest. This stands in stark contrast to other national problems such as cancer, AIDS, and trauma from automobile accidents. There has never been a national focus or effort to improve the outcome from cardiac arrest. Thus, in the 35 years since modern cardiopulmonary resuscitation (CPR) was developed, very little has changed in the therapy for sudden death. Despite the lack of a national mandate (as defined by gobs of money flowing through the National Heart, Lung and Blood Institute), there has been considerable debate about the causes of sudden death, optimal management, and efforts to preserve brain function.

Dr Max Weil, considered by many to be a founding father of critical care medicine, is eminently qualified to edit this book. He and his colleague, Dr Wanchun Tang, have been actively involved in cardiac arrest research and assembled a stellar cast of authors for the individual chapters. The book is designed primarily for physicians, nurses, and emergency medical technicians who are involved in CPR and the management of patients snatched from the jaws of death. In this regard, large portions of the book are relevant to respiratory therapists as well as to physicians and nurses who manage resuscitated patients in intensive care units. Though I don't believe the book will be relevant for the immediate management of cardiac arrest (other textbooks such as *The Textbook of Cardiac Life Support*, published by the American Heart Association, would be more suitable), this book provides background information about cardiac arrest in all its manifestations. Anyone dealing with the resuscitated patient will find much useful information here.

The book has 21 chapters written by a total of 37 authors from throughout the United States and 4 other countries. The first chapter is a history of CPR, followed by discussion of the epidemiology, etiology, incidence, and survival rates. The remaining chapters provide a complete overview of cardiac arrest and its management, including airways, electrical causes, automated external defibrillation, mechanical interventions, pharmacologic therapy, complications and management, central nervous system resuscitation, resuscitation of the infant, emergency medical services, risk of infection, costs and outcomes, training, and ethical and legal considerations. In spite of being a multi-author book, the chapters are reasonably self-contained topics and, thus, there is relatively little overlap between the chapters.

The publisher, WB Saunders, has done a professional job in design and production qualities. The type is readable and the figures and illustrations are clearly reproduced. Frequent headings and subheadings break up the text and allow one to find material quickly using the complete index.

The book's publication date is 1999. Most of the references are from 1996 or earlier. The material seems up to date, with some exceptions; for example, the material on cardiac enzymes in the chapter on acute myocardial infarction is dated. Hardly anyone orders lactate dehydrogenase measurements anymore for diagnosing acute myocardial infarction. Despite such infrequent lapses, the book is a nice blend of the practical and the experimental and provides a comprehensive state-of-the-art view of cardiac arrest and its management.

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Manual of Pediatric Critical Care. Mary Fran Hazinski MSN RN. St Louis: Mosby. 1999. Softcover, illustrated, 688 pages, $42.95.

The needs of patients, families, and loved ones can easily be overlooked when we are confronted with the many crises that occur in the pediatric intensive care unit. Frequently, respiratory therapists focus solely on the ventilatory needs of our patients, while leaving the other matters to the physician and nurse. That's why it is refreshing to see things from a different perspective. This book was not designed to be a comprehensive reference, but, rather, it provides the

reader with core information that can be gleaned in a matter of a few minutes' reading. Its intended audience is the bedside nurse.

This book's chapters are aligned to body systems, allowing easy access to organ-specific information. There is some redundancy of details among the chapters, but if the reader is to glean all that is pertinent to each distinct problem or body system, this type of a presentation makes sense. A useful feature of this text is that at the beginning of each chapter is a list of "*pearls*," which is a means of alerting the reader to important concepts that should be remembered as they read the chapter.

Chapter 1 presents a brief overview of how the child in the critical care unit is different and unique. This chapter attempts to drive home the point that the sick child cannot be treated in the same fashion as an adult. Pediatric patients exhibit unique signs and symptoms that vary, even between pediatric subgroups. We cannot treat pediatric patients as *little adults*.

Chapter 2 presents the psychosocial issues faced in pediatric critical care. Separation anxiety and death are discussed in great detail. Because each subgroup of pediatric patients has a different cognitive level, the author looks at these issues from the aspect of the infant, toddler, preschool child, school-age child, and adolescent. It is fascinating to see how each age processes these issues quite differently.

Chapter 3 reviews the complex issues of how this population of patients can and should be relieved of unnecessary pain and anxiety. Pain assessment is well presented, as are the analgesics, narcotics, sedatives, neuromuscular blockers, and antagonists.

Chapter 4 is a more intensive review of end-of-life issues. Though an earlier chapter did discuss this, more attention is directed at issues faced by the patient's nuclear family. The impact of a child's death is felt differently by all age groups. This chapter skillfully discusses some of these perceptions and methods to work with those affected by the death of a child.

Chapter 5 comprises 205 pages—almost a third of the book—and reviews cardiovascular disorders in great detail. The size of the chapter is an indication that cardiovascular disorders are a frequent and important problem in the critical care unit. Because of the enormity of this chapter, perhaps a separate cardiovascular text would be more appropriate. There is much to digest in this section of the book.

Chapter 6 discusses pulmonary disorders. An attempt is made to present key issues that the bedside nurse may encounter with those who suffer from respiratory compromise. Unfortunately, not enough detail is offered and some questionable recommendations are made. The book states that the outcomes of incentive spirometry can be obtained just as easily by having the child blow bubbles or blow paper cups across a bedside table. That generalization is not correct and is similar to the old philosophy of using blow-gloves and blow-bottles, which are expiratory maneuvers and have been associated with reduced lung volumes. Patients must be coached to take deep and prolonged inhalations, and an incentive spirometer is the best device to do this. Also, the author repeatedly recommends that oxygen be delivered with humidification and heat: this is not accepted practice in all circumstances. On Page 307 the oxygen delivery table indicates that an oxygen tent can provide 24–100% oxygen, whereas a hood can only give 24–40% oxygen. That information is incorrect and, in fact, it is the other way around.

Pediatric asthma is the most common discharge diagnosis in pediatric hospitals, and it is commonly seen in the pediatric intensive care unit. Despite this fact, asthma was addressed on only two pages of the book. Consideration should have been given to a more expanded review of this disease. Some of the recommendations in this section about the dosing of albuterol and classification of asthma were dated and not in compliance with the EPR-2 Expert Panel Report, which, interestingly, wasn't even noted as a reference.[1]

Chapter 7 focuses on chest radiograph interpretation, which is a vitally important subject for the bedside clinician to understand. I especially appreciated seeing a discussion of endotracheal tube placement. The simple flexing or rotation of the patient's neck can move the tube farther into or out of the trachea. Proper tube placement for chest radiography is essential. Often we overlook these points when assessing endotracheal tube placement.

Chapter 8 reviews the many neurological disorders of pediatrics, and this topic is well presented.

Chapter 9, on renal disorders, provides important facts about and indicators of pediatric renal failure and related complications.

Chapter 10 is devoted to gastrointestinal disorders. Fluid and electrolyte balance and its impact on the child are presented in logical order. Table 10–11 is especially useful in its presentation of the etiology, pathophysiology, signs and symptoms, and nursing intervention for each gastrointestinal disease.

Hemologic and oncologic emergencies are reviewed in Chapter 11. This is an area that may be the most foreign to the respiratory therapist.

Chapter 12 is devoted to pediatric trauma. Because of the large numbers of trauma patients in pediatric intensive care units, this section of the book is of immense importance for both the bedside nurse and emergency response field personnel. Mishandling a patient before stabilization in the intensive care unit is likely to result in long-lasting negative sequelae.

The last chapter of the book is a discussion of pediatric burns. This is a subject not often addressed in texts like this, but is certainly needed. A brief but thorough presentation of the subject is made.

I would recommend the **Manual of Pediatric Critical Care** to all pediatric bedside clinicians, including nurses and respiratory therapists, who should use it as a bedside reference and may gain a better understanding of the whole patient picture, beyond just the respiratory aspects. Many pearls of wisdom are presented and deserve the attention of all caregivers. The book is reasonably priced and would be a valuable reference. I found the book's blue font a bit distracting and it took some getting used to, but perhaps that is an aesthetic issue for me and not for other readers.

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REFERENCE

1. National Asthma Education and Prevention Program Expert Panel Report 2: Guidelines for the diagnosis and management of asthma. National Institutes of Health Publication No. 97–4051.

A Guide to Infection Control in the Hospital. An official publication of the International Society for Infectious Diseases. R Wenzel, M Edmond, D Pittet, JM Devaster, T Brewer, A Geddes, and J-P Butzler, Editors. Hamilton, Ontario: BC Decker. 1998. Soft-cover, 182 pages, $24.95.

In the United States approximately 5% of patients admitted to hospital acquire infections. These nosocomial (hospital-acquired) infections increase morbidity and mortality, and cost more than $4.5 billion per year. The Centers for Disease Control and Prevention estimate that infection control programs that include specific prevention components (eg, calculating surgeon-specific surgical site infection rates and providing those data to surgeons) may prevent as many as 32% of nosocomial infections.

In developing countries the problem of nosocomial infection is even more acute, and nosocomial infection rates may exceed 25%. Fortunately, as many as 40% of these potentially serious hospital complications may be prevented with an organized infection control program. Unfortunately, in many developing countries, the practice of infection control is in its infancy, and resources and governmental support may be scarce. The purpose of this booklet is to provide a blueprint for the design and implementation of an infection control program in developing countries where resources may be limited. The benefits of such a program are lower nosocomial infection rates, improved quality of care and outcomes, and reduced costs.

Edited by an international group of physicians well known in infection control research and publication, Drs Wenzel, Edmond, Pittet, Devaster, Brewer, Geddes, and Butzler, the booklet has a total of 45 contributors, also well known international authors and researchers in the field of infection control. The booklet comprises 39 chapters, dealing with various aspects of a comprehensive infection control program. Chapter 1 provides the rationale, justification, and benefits of establishing an infection control program. Subsequent chapters cover the various components of a complete program: handwashing, isolation practices, sterilization and disinfection, key hospital departments, hospital-acquired infections, and key nosocomial pathogens such as *Staphylococcus aureus*, the enterococci, and *Pseudomonas aeruginosa*. The chapters all follow the same format, and succinct paragraphs highlight key issues, known facts, controversial issues, and recommended practices, followed by 2–6 key references. The chapter format allows ready identification of critical factors in establishing or evaluating infection control practices both hospital-wide and in various departments.

A minor distraction of the booklet is that the sequence of chapters is at times disjointed. For example, Chapter 23, "Diphtheria, Tetanus, Pertussis," and Chapter 24, "Measles," are sandwiched between chapters that discuss nosocomial infections or risks, such as pneumonia and blood transfusions/intravenous fluids. Grouping chapters by broad subject headings, such as developing and organizing a program, department-specific issues, the major nosocomial infections, and then specific organisms and diseases would better organize the content.

It is the intention of the editors to revise the booklet every two years, which is a must in the fast-paced, ever-changing field of infection control. This booklet is a fine reference for practitioners struggling to institute standardized infection control practices in health care settings in developing countries. It may also serve as a handy reference to department managers or specialists evaluating specific components of an infection control program in developed countries as well. Although it does not provide the detail or literature reviews found in infection control reference texts, it is a useful handbook of infection control practice.

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Systematic Reviews: Synthesis of Best Evidence for Health Care Decisions. Cynthia Mulrow MD MSc and Deborah Cook MD MSc(Epid), Editors. Philadelphia: American College of Physicians. 1998. Soft-cover, 117 pages, $27.

Systematic reviews are an increasingly popular form of peer-reviewed publication in the medical literature. Many major medical journals are moving away from the review article written by an expert and toward the systematic review that is conducted in a rigorous and reproducible manner. Over the past decade, systematic reviews have addressed important clinical questions affecting respiratory care practitioners and ranging from prophylaxis for acute gastrointestinal bleeding in the intensive care unit, to the prevention of ventilator-associated pneumonia, to the diagnosis of pulmonary embolism. This book, edited by Mulrow and Cook, provides a detailed summary of the science and methods of systematic reviews. It contains 10 chapters that include diverse topics such as where systematic reviews fit within the medical evidence base, how to locate and appraise systematic reviews, and how to use the results in clinical practice, clinical education, and health care policy. The last 4 chapters provide detailed information on how to conduct and report systematic reviews.

This book is most useful for those aspiring to conduct systematic reviews and for those wanting a thorough understanding of the role systematic reviews can play in shaping our health care system. The level of detail in much of the book is more than one needs to be able to read and appraise systematic reviews. However, for those interested in conducting systematic reviews, this book provides an excellent guide for formulating the questions to be addressed by a systematic review, selecting and assessing the studies to be reviewed, and summarizing this information in a way that is most useful to the reader.

The book is brief, relatively succinct, and well organized. The writing styles vary by chapter, but on the whole it reads well and does not require training or background in epidemiology or statistics to understand. The book lends itself to a quick read from cover to cover, and yet the organization is such that the reader can quickly identify the particular area they are interested in and obtain specific information. The chapters each have a clear title and abstract, and the book makes good use of subheadings, tables, and figures.

Systematic reviews have a couple of important limitations. First, they are only useful when there are several studies addressing the same or a very similar question and

when these studies are of sufficient quality and are conducted in a similar manner such that the results can be summarized in a meaningful way. Many important clinical questions do not lend themselves to systematic reviews because there are insufficient studies or the studies available are of insufficient quality. Second, as the authors point out, systematic reviews can aid but never replace sound clinical reasoning. Systematic reviews can summarize and add to the evidence base, but evidence can lead to bad practice if it is applied in an uncritical or insensitive way.

Mulrow and Cook have done an excellent job of achieving one of the goals they established, namely to develop a book that can increase the understanding of the role systematic reviews can play in advancing knowledge. The editors also hope that their book will facilitate the use of systematic reviews and motivate more reviewers to conduct systematic reviews. Their book certainly has that potential because it provides readers with the information they need to use and conduct these reviews, but only time will tell if it achieves these latter two aims.

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CORRECTIONS

In the article, "Influence of Inspiratory Flow Rate, Particle Size, and Airway Caliber on Aerosolized Drug Delivery to the Lung" (Respir Care 2000;45:597-608), the middle initial of the author's name, Myrna B Dolovich PEng was mistakenly printed as A instead of B.

Page 598, line below equation: for "$cm^2 = d^2/4$" read "$cm^2 = \pi\, d^2/4$".

Page 606, second-to-last line of the Summary: for "data" read "outcomes data".

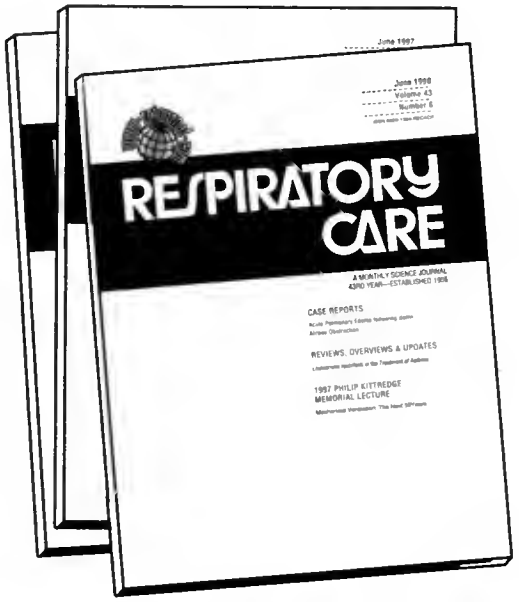
The correction to Prof Dolovich's name also applies to her listing as co-author of the article, "Lung Models: Strengths and Limitations" (Respir Care 2000;45:712-736), to her listing in the Conference Faculty and Writing Committee on Page 586, and to her listing as a co-author of the "Consensus Statement: Aerosols and Delivery Devices" (Respir Care 2000;45:589-596).

We regret the error and the omissions.

In the editorial, "Bronchodilation in Mechanically Ventilated Patients: How Much Is Enough and How Best to Deliver?" (Respir Care 2000;45:815-816), an erroneous medication dosage was printed.

Page 816, left-hand column, third paragraph,4th line down: for "up to 15 mg/h of aerosolized albuterol" read "up to 7.5 mg/h of aerosolized albuterol".

We regret the error.



CRCE through the Journal—2000

CRCE through the Journal, a program for American Association for Respiratory Care (AARC) members to gain credit for continuing education, is now in its eleventh year. By reading RESPIRATORY CARE–the science journal for respiratory care professionals—and completing this examination, AARC members may earn credit for continuing education.*

This 50-item, multiple-choice examination is based on papers published from July 1999 through June 2000 in RESPIRATORY CARE. The issue and page numbers of the paper on which a question is based are shown in brackets following the question. You may consult the cited paper; however, we encourage you to read the paper in its entirety before answering the question. Choose the single most-correct answer, and mark the answer sheet, which is located following Page 1144.

Mail your completed answer sheet by October 31, 2000. Answer sheets postmarked after October 31, 2000 will not be processed. The Answer Key for CRCE through the Journal will be published in the November issue of RESPIRATORY CARE. AARC members can access their CRCE transcripts via the AARC's Web site: http://www.aarc.org. Your CRCE through the Journal results will be available as soon as your answer sheet is scored and the score posted to your transcript.

We are indebted to Phillip D Hoberty EdD RRT, Assistant Professor and Director of Clinical Education, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, Columbus, Ohio, for coordinating the examination and to his co-authors: F Herbert Douce MS RRT RPFT, Associate Professor and Director; Georgianna G Sergakis BS RRT, Graduate Teaching Assistant; and Sarah Varekojis MS RRT, Lecturer, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, Columbus, Ohio; and Timothy B Opt'Holt EdD RRT, Associate Professor, Department of Cardiorespiratory Care, University of South Alabama, Mobile, Alabama.

*The acceptance of these credits for the fulfillment of license-mandated continuing education is dictated solely by the licensure law of each individual state.

QUESTIONS:

Please follow the instructions on the previous page, and record your answers on the perforated form provided following Page 1144.

1. According to Hess, there is evidence that use of the closed suction system:

   a. is inferior to conventional suction technique in clearing secretions.
   b. must include daily equipment changes.
   c. leads to inadequate oxygenation during suctioning.
   d. may result in cost savings over conventional suctioning.

   [July 1999;44(7):759-776]

2. According to Watson, which is the correct approach to dealing with a difficult direct laryngoscopy (DL) for intubation?

   a. When intubation difficulty is predicted, alternative equipment and individuals with special skills should be assembled prior to attempting DL.
   b. Alternative equipment should be stored only in the operating room.
   c. When unexpected difficulty is encountered, attempts should be limited to about 6-8 attempts.
   d. If the first attempt at DL fails, the least skilled individual should make the next attempt.

   [July 1999;44(7):777-798]

3. According to Campbell, which statement is NOT true concerning extubation and reintubation?

   a. Patient readiness for extubation and possible reintubation is simple to predict.
   b. Clinicians should diligently monitor patients during the postextubation period.
   c. Reintubation is associated with many reported complications and increased mortality and morbidity.
   d. Reintubation rates should be monitored in each institution and within different care areas.

   [July 1999;44(7):799-806]

4. According to Heffner, which statement is correct concerning making the decision to perform a tracheotomy?

   a. There is an ideal time for tracheotomy, universally applicable to all critically ill patients.
   b. After 2-3 weeks of intubation, tracheotomy is always indicated.
   c. When considering the possible benefits, some ventilated patients may never become candidates for tracheotomy.
   d. Patients' and patient families' perspectives on the relative desirability of tracheotomy should be ignored.

   [July 1999;44(7):807-819]

5. According to Stauffer, which complication is NOT an inevitable consequence of tracheal intubation?

   a. ciliostasis
   b. mild edema at the cuff site
   c. hyperemia
   d. mucosal ulceration

   [July1999;44(7):828-844]

6. According to Branson et al, an active heat and moisture exchanger (AHME) incorporates which of the following components in addition to those found in a hygroscopic heat and moisture exchanger (HHME)?

   a. a wick onto which water is pumped
   b. a hygrometer
   c. a small-volume ultrasonic nebulizer and water feed line
   d. a control for adjusting inhaled gas temperature

   [August 1999;44(8)912-917]

7. Branson et al concluded that the active heat and moisture exchanger:

   a. humidified as well as heated humidifiers with heated wire circuits.
   b. uses more water than a traditional heated

humidifier.
c. had more condensate than the heated wire circuit.
d. had less dead space than the traditional heated humidifier.
[August 1999;44(8):912-917]

8. In an animal model by Takahashi et al, the greatest change in P_{aCO_2} as a result of tracheal gas insufflation (TGI) and/or exsufflation (TGE) occurred during:

a. TGI throughout inspiration and TGE throughout expiration.
b. TGI only, throughout inspiration.
c. TGE only, throughout expiration.
d. TGE for the first half of expiration and TGI for the second half of expiration.
[August 1999;44(8):918-924]

9. According to the study of demand oxygen delivery systems by Bliss et al, one may conclude that:

a. The F_{IO_2} delivered by these systems is equivalent.
b. Demand oxygen delivery systems deliver an F_{IO_2} more consistent than continuous oxygen systems as ventilatory rate increases.
c. The oxygen delivery flow profiles for various demand oxygen delivery systems are equivalent.
d. The oxygen use efficiency for various demand oxygen delivery systems are equivalent.
[August 1999;44(8):925-929]

10. In the study of demand oxygen delivery systems by Bliss et al, what factor accounted for the variable F_{IO_2} delivered by demand-type demand oxygen delivery systems versus continuous flow oxygen?

a. the dilution effect
b. the pooling effect
c. the timing of oxygen delivery
d. the flow of oxygen through the device
[August 1999;44(8):925-929]

11. According to the study by Leidy and Knebel, the Functional Performance Inventory includes measurement of all of the following EXCEPT:

a. household maintenance.
b. physical exercise.
c. pulmonary function.
d. social activities.
[August 1999;44(8): 932-939]

12. Which of the following is NOT recommended by MacMahon to reduce the number of potential pitfalls in the interpretation of portable bedside radiography?

a. awareness of technical issues
b. correct positioning of the patient
c. standardization of techniques
d. horizontal beam projection
[September 1999; 44(9):1018-1032]

13. According to Kazerooni and Cascade, which of the following is FALSE when distinguishing cardiogenic edema from other causes of lung opacity in the chest radiograph?

a. Postoperative atelectasis is often described as "butterfly" in appearance.
b. Diffuse atelectasis is often recognized by secondary signs of volume loss.
c. The hallmark of pneumonia is airspace consolidation accompanied by air-bronchograms.
d. Cardiogenic pulmonary edema generally changes with positioning.
[September 1999; 44(9):1033-1043]

14. According to Collins, which of the following is NOT a chest radiograph sign of traumatic aortic injury?

a. pulmonary contusion
b. hemothorax
c. tracheal shift to the left
d. widening of the mediastinum
[September 1999; 44(9):1044-1063]

15. Gross describes which of the following radiographic interpretations as correct ETT placement in a neonate?

a. ETT tip appears to be positioned at the level of the carina.
b. ETT tip projects below the level of the carina.
c. ETT tip projects just above the medial clavicle ends.
d. ETT tip appears to be positioned 2 cm above the level of the carina.
[September 1999; 44(9):1095-1126]

16. According to Miller, which of the following is NOT a clinical indication for computed tomography?

a. characterization of pleural effusions
b. daily assessment of clinical status
c. staging of thoracic malignancies
d. evaluation of sepsis of unknown cause
[September 1999; 44(9):1127-1136]

17. Which brand of adult ventilator did Blanch demonstrate to have the lowest incidence of operator errors per hour of use?

a. Bird VIP
b. Bird 6400 ST
c. Bird 8400 STi
d. Mallincrodt Nellcor Puritan Bennett 7200 ae
[October 1999;44(10):1183-1191]

18. Ozgun et al identified 4 indications for placing indwelling arterial catheters and developed a protocol for patients admitted to a medical ICU. What was an outcome of following the protocol for the 8 month study period?

a. Number of ABGs was reduced.
b. Catheter placements were reduced.
c. ICU length of stay was increased.
d. Mechanical ventilator hours were increased.
[October 1999;44(10):1193-1197]

19. Punjabi reported that studying the duration of time between occurrences of events, such as time between ventilator malfunctions, is an example of which analytical method?

a. outcomes research
b. survival analysis
c. regression analysis
d. uncensored observations
[October 1999;44(10):1198-1202]

20. Which statement about device selection for bronchodilator resuscitation in the emergency department is consistent with recommendations made by Fink and Dhand?

a. The pMDI/HC is equivalent to nebulizer therapy for treatment of infants, children and adults with moderate to severe asthma.
b. Patients with severe asthma should use DPI due to decreased flows required to actuate the devices.
c. A pMDI/HC is often selected due to decreased cost when compared to a nebulizer when used in the hospital.
d. The ultrasonic nebulizer is more effective than the pneumatic nebulizer for the treatment of severe asthma.
[November 1999; 44(11):1353-1374]

21. According to MacIntyre et al, which statement is CORRECT concerning the use of automated rotational therapy for prevention of respiratory complications during mechanical ventilation?

a. Automated lateral rotational beds were less effective than standard ICU patient turning strategies.
b. Standard ICU patient turning procedures were more effective at prevention of urinary tract infections.
c. Anxiety was noted to be greater in patients turned using standard ICU procedures.
d. No differences in the prevention of respiratory complications were found between the two strategies.
[December 1999; 44(12):1447-1451]

22. In a study by Schmaling et al, which variable was found to be a significant predictor of the absence of bronchial hyperresponsiveness in patients undergoing methacholine inhalation challenge?

a. age < 18 years
b. reversible airflow obstruction
c. anxiety symptoms related to social situations
d. patient was an ex-smoker
[December 1999; 44(12):1452-1457]

23. According to Carella et al, which of the following parameters did NOT change following weight loss in a study of obese women?

    a. TGV
    b. resting oxygen consumption
    c. ERV
    d. IC
    [December 1999; 44(12):1458-1464]

24. Which of the following conclusions is TRUE concerning the activation of MDI spacer devices according to data reported by Foss and Keppel?

    a. Respirable dose from each device was greater in the out-of-phase case.
    b. Timing and device design have great impact on the amount of drug delivered to the patient.
    c. MDI spacer devices equipped with one-way valves were less effective at maintaining the drug plume.
    d. Drug delivery is entirely effort dependent.
    [December 1999; 44(12):1474-1485]

25. According to Pierson, which of the following is the most common mechanism for hypoxemia in patients with chronic pulmonary disease?

    a. mismatching of ventilation and perfusion
    b. right-to-left intrapulmonary shunting
    c. diffusion impairment
    d. low inspired P_{O_2}
    [January 2000: 45(1):39-51]

26. Which of the following is NOT described by Benditt as a form of respiratory system oxygen toxicity?

    a. absorption atelectasis
    b. acute parenchymal lung injury
    c. chronic parenchymal lung injury
    d. chronic hypercapnia
    [January 2000: 45(1):54-61]

27. Hess describes all but which of the following as a potential source of inaccuracy in pulse oximetry measurements?

    a. dyshemoglobinemias
    b. increased perfusion
    c. increased skin pigmentation
    d. anemia
    [January 2000: 45(1):65-80]

28. According to McCoy, which of the following is NOT an option for continuous-flow oxygen conservation?

    a. transtracheal oxygen
    b. reservoir devices
    c. oxygen dilution
    d. titrating to the lowest flow needed per activity
    [January 2000: 45(1):95-103]

29. Criner states that long-term oxygen therapy is indicated for all but which of the following?

    a. stable disease with $P_{aO_2} \leq 55$ mm Hg ($S_{aO_2} \leq 88\%$)
    b. P_{aO_2} 55–59 mm Hg with signs of tissue hypoxia
    c. desaturation during sleep or exercise
    d. normoxemic patients with dyspnea
    [January 2000: 45(1):105-118]

30. According to Wedzicha, what effect does long-term oxygen therapy have on health status?

    a. great improvement
    b. slight improvement
    c. little effect
    d. worsening effect
    [January 2000: 45(1):119-124]

31. Which of the following is TRUE regarding the use of positive pressure nasal ventilation in combination with long-term oxygen therapy in stable COPD patients with hypercapnia, according to Wedzicha?

    a. controls hyperventilation
    b. does not improve quality of life
    c. improves daytime ABGs and sleep
    d. increases number of exacerbations
    [February 2000: 45(2):178-185]

32. According to O'Donohue and Bowman, what percentage of symptomatic COPD patients who are not hypoxemic while awake experience

nocturnal oxyhemoglobin desaturation?

a. 45%
b. 50%
c. 60%
d. 80%

[February 2000: 45(2):188-191]

33. MacIntyre states that the use of supplemental oxygen during exercise will do which of the following?

a. increase exercise ventilation
b. improve hypoxemia and exercise impairment
c. increase air trapping in obstructive disease patients
d. exacerbate dyspnea

[February 2000: 45(2):194-200]

34. According to Stoller, it is important to evaluate air travelers with chronic pulmonary disease for their in-flight supplemental oxygen needs due to the

a. possibility of altitude-induced adverse events during flight.
b. lack of environmental stresses of commercial air travel.
c. hyperbaric conditions experienced during commercial air travel.
d. transient increase in P_{O_2} accompanying assent to higher altitudes.

[February 2000: 45(2):214-221]

35. Which of the following design features of central oxygen supply systems are NOT specifically recommended by Stoller et al?

a. installation of a redundant central oxygen supply system
b. prominent labeling and shielding of oxygen supply lines
c. a remotely located, back-up supply vessel with separate hospital feed lines
d. ample valves to assist in locating leaks in the system

[March 2000;45(3):300-305]

36. A sedated and paralyzed patient is on volume controlled mechanical ventilation; humidification is being provided by a heat and moisture exchanger with a dead space of 90 mL. According to Campbell et al, which of the following actions may correct an increase in P_{aCO_2} in this patient?

a. Increase the level of pressure support ventilation.
b. Place an additional heat and moisture exchanger in series.
c. Increase the trigger sensitivity.
d. Increase the set ventilator rate.

[March 2000;45(3):306-312]

37. Which of the following conclusions is FALSE according to the nebulizer system data reported by Piper?

a. Total treatment time among the three nebulizers was not significantly different.
b. The amount of medication delivered by the Aero Tee was greater than the other two nebulizer systems.
c. The Circulaire system delivered the largest aerosol particles.
d. The lowest rebreathed volume per breath was measured using the Circulaire system.

[March 2000;45(3):313-319]

38. According to Rau and Torniainen, modifying the aerosol MDI delivery system in which of the following ways will increase the total dose of albuterol?

a. addition of a positive expiratory pressure device
b. the use of albuterol with a chlorofluorocarbon propellant
c. the use of a hydrofluoroalkane propellant
d. none of the modifications tested

[March 2000;45(3):320-326]

39. According to Adams's study of the performance of bi-level pressure ventilators, patients with increased airways resistance and increased lung compliance, such as those with COPD, may not tolerate noninvasive positive pressure ventilation because

a. auto-PEEP develops.
b. inspiratory time is reduced.
c. ventilator peak flow increases.
d. delivered tidal volume is too constant.

[April 2000;45(4):390-400]

40. A hospitalized patient with COPD qualifies for continuous home oxygen therapy upon discharge. When the patient is reevaluated by a pulmonary physician for continuing oxygen need, what does Oba report as commonly occurring?

a. The initial prescription for oxygen therapy is continued.
b. The initial prescription for oxygen therapy is changed to nocturnal use only.
c. The reevaluation includes arterial blood gas analysis or pulse oximetry at rest while breathing room air.
d. The reevaluation includes routine pulmonary function testing.

[April 2000;45(4):401-406]

41. According to Vilke et al, when a patient is unable to stand for spirometric testing, which alternate testing position will produce the best results for normal men?

a. sitting
b. supine
c. dorsal
d. prone

[April 2000;45(4):407-410]

42. According to Tobin, when predicting the outcome of a weaning trial from continuous mechanical ventilation, what measurement provides the most accurate prediction?

a. MIP @ FRC
b. f/ V_T ratio
c. V_D/V_T ratio
d. CROP index

[April 2000;45(4):417-431]

43. What is the reported result when a respiratory therapist arterial line placement service is initiated in a university hospital intensive care unit?

a. Initial arterial cannulation success rate was 80%.
b. Frequent complications occurred with dorsalis pedis cannulations.
c. Cannulations on second attempts by the same therapist were always successful.
d. Patients were routinely subjected to multiple cannulation attempts.

[May 2000;45(5):482-485]

44. Fink and Dhand reviewed the literature on bronchodilator resuscitation for severe asthma exacerbations in the emergency department. One of their conclusions was:

a. The manufacturer's standard recommended dose should be followed in the emergency setting.
b. Using low-dose high-frequency therapy results in the lowest rate of hospital admissions.
c. Patients with severe asthma benefit most from beta agonists combined with anticholinergic drugs.
d. Metered dose inhalers with holding chambers should be discouraged in the emergency setting.

[May 2000;45(5):497-512]

45. In 1999, the National Lung Health Education Program (NLHEP) made recommendations for spirometry in medical offices. According to these recommendations, primary care providers should

a. use only currently available diagnostic spirometers.
b. perform spirometry on all smokers, 45 years and older.
c. obtain 20 hours of specialized training in spirometrics.
d. refer to a hospital pulmonary laboratory all individuals with respiratory symptoms.

[May 2000;45(5):513-530]

46. In assessing the benefit of an aerosolized drug system, the authors of the Consensus Statement believe that third party payers are likely to demand improvement in which of the following?

a. gas exchange
b. health care costs
c. pulmonary function
d. mucus characteristic/volume
[June 2000;45(6):589-596]

47. According to Hess, what is the most important characteristic of nebulizer performance?

a. the respirable dose provided for the patient
b. nebulization time
c. cost of the equipment
d. requirements for cleaning and sterilizing
[June 2000;45(6):609-622]

48. Fink does NOT recommend which component of patient training in teaching proper use of the pressurized metered-dose inhaler (pMDI)?

a. spending about 10-30 minutes in patient instruction
b. including demonstration, practice, and confirmation of patient performance
c. teaching that the pMDI should always be stored with the cap on
d. teaching that the pMDI should be floated in water as a means of determining when the canister is empty
[June 2000;45(6):623-635]

49. Which recommendation is consistent with Dhand's review on delivering an aerosol via an MDI with an artificial airway?

a. Place the nebulizer as close to the endotracheal tube as possible.
b. Set a high inspiratory flow on the ventilator during nebulization.
c. Use an endotracheal tube that is as small as possible.
d. Use the spacer in-line with the MDI.
[June 2000;45(6):636-645]

50. Adjustment of aerosol regimen to optimize delivery via MDI requires attention to which of the following?

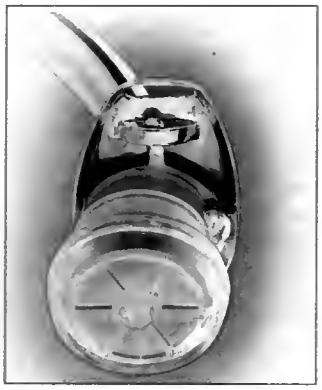
1. proper patient technique
2. choice of aerosol spacer
3. choice of patient interface
4. spacer cleaning
5. consideration of the medicine to be aerosolized

a. 1 and 2 only
b. 1, 2 and 4 only
c. 1, 2, 3 and 4 only
d. 1, 2, 3, 4, and 5
[June 2000;45(6):646-651]

Make sure to double check your work and to mark the answer sheet clearly. Mail your completed answer sheet by October 31, 2000 to

CRCE through the Journal
11030 Ables Lane
Dallas TX 75229-4593

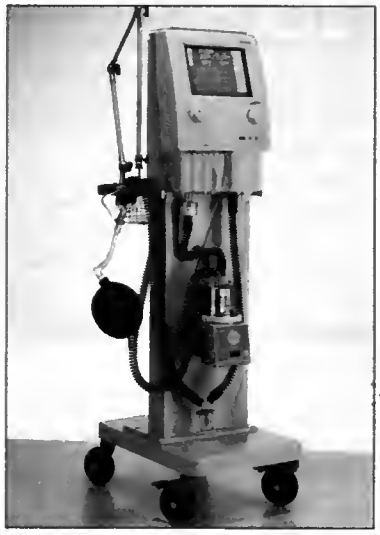
Look for the answers in the November 2000 issue of RESPIRATORY CARE.



Tracheostomy Speaking Valve. Passy-Muir Inc introduces their new PMV 2020 (clear; 15 mm I.D./23mm O.D.) tracheostomy speaking valve. The company describes the device as a light-weight, one-way, closed position "no leak" tracheostomy speaking valve that attaches to the Pilling Weck metal Jackson Improved trach tubes (sizes 4-6 or equivalent) with the use of the PMA 2020-S Adapter. According to Passy-Muir, the device can be used on the adult, pediatric, and neonatal Bivona non-foam filled cuffed tracheostomy tubes currently on the market. The PMV 2020 (clear) is intended for use by both short-term and long-term adult, pediatric, and neonatal non-ventilator dependent tracheostomized patients. For more information from Passy-Muir, circle number 170 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Portable Volume Ventilator. Respironics has released a new portable volume ventilator designed to help patients in many settings who require ventilatory support, but not oxygen. According to the company, this device is ideal for subacute care, nursing care and at-home settings and is featured within Respironics new Respiratory and Failure Management Program. Respironics says that the PLV-102b has a broader and more accurate volume range than most other ventilators and that its updated color scheme allows it to blend well into its surroundings. For more information from Respironics, circle number 171 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Sleep Study System. Grass-Telefactor, a product group of Astro-Med Inc, announces the release of their newly designed polysomnograph (PSG). The company says the Aurora™ PSG system combines compact, high-performance amplifiers with a powerful, easy-to-use recording and analysis software program called Gamma. According to Grass, there are 10 DC channels for auxiliary sensors and instruments and the 32-channel amplifier system can record and interface with all bioelectric PSG signals. For more information from the Grass-Telefactor group of Astro-Med Inc, circle number 172 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/



Ventilator Cleared by FDA for Infant Use. Hamilton Medical Inc has received 510(k) FDA clearance for the Galileo Ventilator for use in infant applications. According to Hamilton, this new application builds on the company's proximal airway technology, providing precise measurement of pressure, volume, and flow for the small infant, with a new minimum deadspace flow sensor. The company says this feature, along with the device's Adaptive Pressure Technology provides the clinician with advanced programmable philosophies or operation, including minimum pressure strategies to optimize ventilatory support for infants through adults. For more information from Hamilton Medical, circle number 173 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

www.rcjournal.com

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

September 14-15–*Pittsburgh, Pennsylvania*

The PSRC Western Regional Pulmonary Conference will be held in conjunction with The Greater Pittsburgh Sleep Professionals at the Sheraton Station Square. The conference features education presentations on management, critical care, sleep diagnostics, pulmonary rehabilitation, the physician forum, and the GPSP sleep tract. **Contact:** Debbie Logan at (800) 545-4663, ext 112.

September 20-22—*Rochester, Minnesota*

The Minnesota Society for Respiratory Care host their 31st Annual Fall State Conference — "Too Hot to Handle." **Contact:** For more information, contact Laurie Tomaszewski at (651) 232-1922, Carolyn Dunow at **dunowc@fhpcare.com**, or Carl Mottram at **mottram.carl@mayo.edu**.

September 27–29—*Hot Springs, Arkansas*

The ASRC will hold its 29th Annual State Meeting and Education Seminar at the Austin Hotel and Hot Springs Convention Center. Speakers include AARC President Garry Kauffman, Vijay Deshpande, Dr Mark Heulitt, Dr Teofilo Lee-Choing, Harold Davis, Jeff Standridge, Lela Parish-Cooper, Shelley Dedman, James Lisenbey, John Campbell, and Theresa Gramlich. Topics will include ventilation in Y2K, home health, pulmonary medicine, asthma, and patient/practitioner rights. National vendors will present an exhibit of the latest technology. Contact John W Lindsey at (870) 541-7606 or **jlindsey@ahecpb.uams.edu**. Or visit **www.arksrc.org**.

October 6—*Cincinnati, Ohio*

The AARC is presenting three postgraduate courses immediately prior to the annual Congress. "Mechanical Ventilation" will include lung injury, managing airway obstruction, weaning, closed loop/dual control modes, and noninvasive ventilation. "Making Protocols Work in Your Institution" will cover rationale, legal aspects, and use in the ICU. "Developing and Enhancing Pulmonary Rehabilitation Services" addresses staffing, patient screening, reimbursement, and marketing. All courses are approved for continuing education credits, and attendance space is limited. **Contact:** For more information, see the Congress Program in the *AARC Times* July issue or call (972) 243-2272.

October 11-13–*Bossier City, Louisiana*

The LSRC's second annual Fall Convention will be held at the Isle of Capri Hotel and Casino with numerous national speakers and exhibitors. Ten CEUs will be available. **Contact:** For more information or booth reservations, call Shonda Houston at (318) 226-0555 or e-mail **sparta@lsumc.edu**.

November 10–12—*Ithaca, New York*

The NYSSRC and the ALACNY will host the Northeast Pulmonary Teaching Conference at Cornell University. Faculty includes Jim Fink, Dean Hess, Bob Kacmarek, Neil MacIntyre, David Pierson, and more. Sessions examine acute and subacute respiratory care, RT management, NIPPV, pediatrics, and asthma. Exhibits, job fair, and family-friendly activities. **Contact:** For more information, call Patricia Kuhl at (315) 422-6142 or visit **www.nepulmonaryconf.com**.

Other Meetings

September 26–29—*TechShop 2000*

If you are a polysomnographic technician seeking to enhance your professional skills, you need information about TechShop 2000. TechShop 2000 is a four-day introductory to intermediate level, hands-on, intensive training for polysomnographic technicians that covers patient preparation, set-up, scoring, troubleshooting, artifact recognition, and much more. Beginning Tuesday, Sept 26, and ending with Keynote Symposium Sept 29, TechShop 2000 is taught at one of the world's premier sleep disorders centers, Ohio Sleep Medicine Institute, under the medical direction of board-certified sleep specialist Dr Helmut Schmidt. For a detailed course outline, contact Crystal or Brian at (614) 792-7632 or e-mail **sleepohio@aol.com**.

Practical Spirometry Certification Course

Two-day hands-on NIOSH-approved course presented by Mayo Pulmonary Services: Sept 29–30 in Chicago IL; and Nov 9–10 in Rochester MN. NIOSH approval #57. Approved by AAOHN for 15.6 contact hours. **Contact:** For further details, call (800) 533-1653.

March 21–24, 2001—*Big Sky, Montana*

The American Lung Association of the Northern Rockies will host their 20th annual Big Sky Pulmonary & Critical Care Medicine Conference at the Big Sky Ski Resort. This multidisciplinary review and update for all health professionals interested in pulmonary and critical care medicine will offer 15 hours of CME credit. **Contact:** For more information, call (406) 442-6556 or e-mail **alamtwy@aol.com**.

call for abstracts

RESPIRATORY CARE • OPEN FORUM 2001

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in San Antonio, Texas, December 1-4, 2001. Accepted abstracts will be published in the October 2001 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions*. Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocal valuation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Times makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in one paragraph on a clean sheet of paper, using margins set so that the abstract will fit into a box no bigger than 18.8 cm (7.4") by 13.9 cm (5.5"), as shown on the reverse of this page.* Insert only one letter space between sentences. Text submission on diskette is allowed but must be accompanied by a hard copy. *Data may be submitted in table form, and simple figures may be included provided they fit within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to Linda Barcus at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by May 31, 2001 will be reviewed and the authors notified *by letter only* to be mailed by June 15, 2001. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (July 17, 2001).

Final Deadline. The mandatory Final Deadline is July 17, 2001 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by September 1, 2001.

Mailing Instructions. Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

2001 RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

submit your OPEN FORUM abstract electronically
visit www.rcjournal.com

RESPIRATORY CARE OPEN FORUM 2001 Abstract Form

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.**
5. All text and the table, or figure, must fit into the rectangle shown. (Use only 1 clear, concise table or figure.)
6. Submit 2 clean copies.

Mail original & 1 photocopy (along with postage-paid postcard) to

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Early deadline is May 31, 2001 (postmark)

Final deadline is July 17, 2001 (postmark)

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American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classificatians in the right-hand column, then complete the applicable section. All informatian requested belaw must be provided, except where indicated as optianal. See other side far mare infarmation and fee schedule. Please sign and date application an reverse side and type or print clearly. Processing of applicatian takes approximately 15 days.

- ☐ Active
  Assaciate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name _____

First Name _____

Social Security No. _____

Home Address _____

City _____

State _____ Zip __ __ __ __ __ – __ __ __ __

Phone No. (_____) _____

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educatar
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Hame Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

Type of Business

- ☐ Haspital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educatianal Institutian
- ☐ Manufacturer ar supplier
- ☐ Other, specify _____

Date of Birth (aptianal) _____ Sex (aptianal) _____

U.S. Citizen? _____ Yes _____ No

Have you ever been a member af the AARC? _____

If sa, when? Fram _____ ta _____

Preferred mailing address: ☐ Hame ☐ Business

For affice use anly

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories ar was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place af Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Medical Director/Medical Spansar _____

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals wha hold a position related to respiratory care but da nat meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whase primary occupation is directly ar indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment ar supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place af Emplayment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members da not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion af your respiratory care educatian, continuing education credits may be pursued upon your reclassification ta Active or Associate Member.

School/RC Program _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify _____

Expected Date of Graduation *(REQUIRED INFORMATION)*

Manth _____ Year _____

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

American Association for Respiratory Care MEMBERSHIP APPLICATION

Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature _____

Date _____

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associate (Foreign)	$102.50
☐ Special	$ 87.50
☐ Student	$ 45.00
TOTAL	$_____

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00
TOTAL	$_____

GRAND TOTAL = Membership Fee plus optional sections $_____

- ☐ Total Amount Enclosed/Charged $_____
- ☐ Please charge my dues (see below)

To charge your dues, complete the following:

- ☐ MasterCard
- ☐ Visa

Card Number

_ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _

Card Expires _____/_____

Signature _____

Mail application and appropriate fees to:

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • [972] 243-2272 • Fax [972] 484-2720

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ___ of ___

Form Approved: OMB No. 0910-0291 Expires: 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Cere)

Triage unit sequence #

A. Patient information

1. Patient identifier
In confidence

2. Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4. Weight
___ lbs
or
___ kgs

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)
☐ death ___ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other: ___

3. Date of event (mo/day/yr)

4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other: ___

5. Expiration date (mo/day/yr)

6.
model # ___
catalog # ___
serial # ___
lot # ___
other # ___

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address

phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

DA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

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9	○	○	○	○	○	○	○

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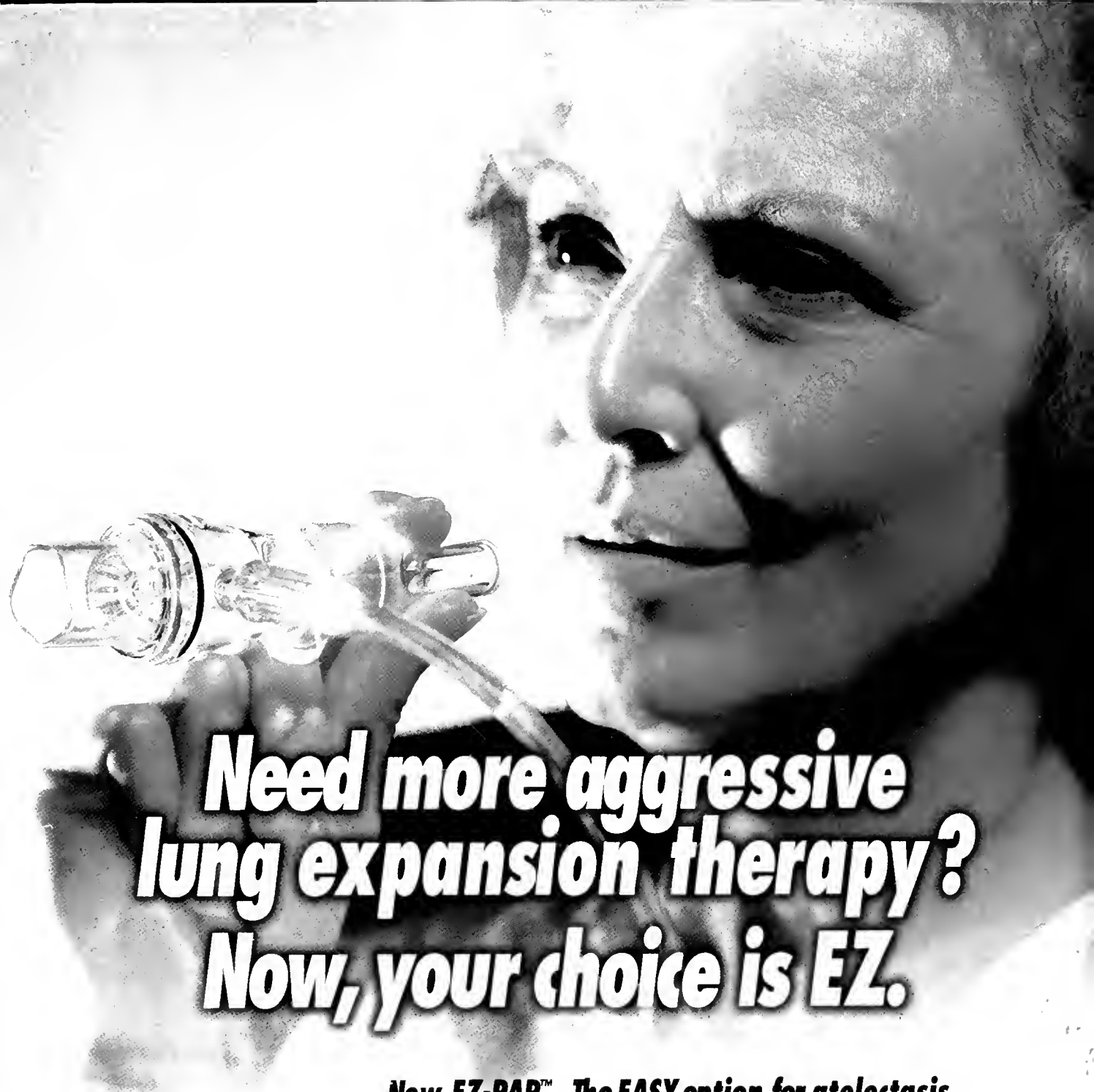

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